# THE MONOFIXATION SYNDROME

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AS THE EVALUATION of strabismus therapy became more critical, attention was focussed upon a relatively large group of patients who had a very small residual deviation. This group had attracted particular attention because, in addition to the consistent findings of a deviation measuring 8 prism diopters or less, there was a suppression area within the deviated eye that prevented diplopia and good fusional vergence amplitudes.

Further interest was stimulated when it became apparent that some patients with very small deviations had no history of strabismus. Anisometropia was identified early as a frequently associated factor in the non-strabismic cases. But even more interesting was the discovery that this disorder was found in some of the population free of strabismus and anisometropia. After it was noted that the common denominator in all these patients with a small deviation was their small facultative central scotoma within the visual field of one eye, binocular perimetry studies on a control series of patients with straight eyes revealed that some of these patients also had a central scotoma. Lastly, it was recognized that the rare patient with a unilateral macular lesion, having straight eyes and peripheral fusion, has the organic counterpart to the unilateral functional facultative central scotoma described above.

Consequently, a specific ophthalmologic entity, either with or without a small deviation, characterized by monofixation due to the central scotoma precluding bifixation and a very active peripheral binocular vision constitutes the monofixation syndrome. The purpose of this paper is to bring together the loose and disorganized components of this syndrome, developing an over-all concept about this disorder that occurs within binocular single vision. A study of 100 patients with the monofixation syndrome will be presented.

## REVIEW OF THE LITERATURE

Initial interest in the monofixation syndrome came by way of the small angle deviations. Jampolsky[21] first intimated, in 1951, something

special about the small angle esodeviated cases and alluded to Pugh's[48] descriptive term for the small deviation as esophoria with retinal slip. Jampolsky briefly mentioned the suppression in the central visual areas in esophoria patients with retinal slip, but claimed that it disappears when the major amblyoscope's tubes are moved to the fusion-free position (the objective angle or position of rest). This does not actually happen in the monofixation syndrome, but it is significant that he was the first to draw attention to the central suppression and peripheral fusion in the small angle deviations. Also in 1951, Gittoes-Davies[14] reiterated Pugh's view regarding the lack of success in orthoptically treating these small deviations, and in 1953[15] she identified these cases as esophoria with fixation disparity. Bryer,[6] in 1953, referred to these patients as flicker cases because the cover test revealed the small "flick" as the deviated eye assumed fixation. Cashell,[12] in 1954, applied the name fixational disparity to the small "flick" cases. In 1953, Levinge[29] and in 1955, Boardman[5] discussed these cases as fixation disparity. Also in 1955, Lyle and Foley[32] described the small angle deviations with peripheral fusion as subnormal binocular vision.

Jampolsky,[20] in 1956, differentiated the small angle esodeviations into small angle esotropia and convergent fixation disparity, establishing many of the features of the monofixation syndrome as we know it today. He described how some of the convergent fixation disparity patients have a greater alternate cover measurement than cover-uncover and emphasized that this is diagnostic of the disorder, if present. Apparently by 1956 he had formulated a firmer concept than in 1951[21] about the suppression within the central retinal area in one eye of these patients, using this as an explanation for solving the diplopia that the minimal deviation causes for the object of regard. He further reasoned that the peripheral portion of Panum's fusional space is sufficiently large to permit fusion with normal retinal correspondence (NRC). His opinion regarding lack of success by orthoptic treatment for these patients is clearly stated. Two empirical generalizations were reported, both of which have been repudiated: "From a practical viewpoint the following empirical correlations have been found to be clinically useful. Patients with a small residual convergent deviation following the optical correction of a significantly high hypermetropia usually are found to have a convergent fixation disparity. On the other hand, patients with a postoperative residual small degree convergent deviation usually are found to have a small angle esotropia," and "It is of prognostic interest to realize that patients with convergent fixation disparity frequently overcome the anomaly with

time alone and develop stable fusion, while patients with small degree esotropia remain so permanently."

Jampolsky also noted the paucity of small angle exodeviations compared with the frequent cases of convergent fixation disparity. In 1957 Jampolsky and associates[23] suggested that this might be related to their findings that the quantity of esofixation and exofixation disparity do not accompany the quantity of distant horizontal heterophoria in a similar manner. The esofixation disparity quantity varies according to the amount of distant esophoria, but the exofixation disparity remains essentially constant throughout the entire range of exophoria quantities at distance. The name "convergent fixation disparity" used by Jampolsky in 1956[20] describes a different entity than "fixation disparity" used by Jampolsky and associates[23] in 1957. Fixation disparity was used by Ogle and associates[39] in 1949 to designate the inexactness of the intersecting visual axes at the point of regard that occurs while bifoveally fixating.* Despite the sustained research and continued reporting of Ogle and his collaborators[33,35–38] on the subject of fixation disparity, the term fixation disparity was gradually being usurped by the clinician to describe the monofixation syndrome, a pathologic condition, rather than the normal bifoveal fusion it was intended to describe. A separate report on what fixation disparity was and was not was required by Ogle in 1958[34] before thwarting the inadvertent confiscation of this term by the clinician. In 1962 Jampolsky[22] referred to the monofixation syndrome as fusion disparity and defended this term – "Fusion disparity is heterophoria in which there is not exact bifoveal fixation – hence the term is used to imply fusion (phoria) with a disparity in bifixation." He continues, "Some confusion exists relative to the use of the term fixation disparity. Some authors prefer to reserve this term for the physiologic fixation disparity which occurs in normal persons during apparent bifoveal fixation fusion. The small amount of physiologic fixation disparity is detectable only by delicate laboratory methods. Some patients with a small degree of esodeviation exhibit larger, grossly observable amounts of fixation disparity, and since they fulfill the definition criteria, are also

*Verhoeff[51] objects to the term "bifoveal fixation" for designating "binocular fixation" or "fusion" because it suggests that only the foveas are participating. He shortened "binocular fixation" to "bifixation." I use "bifixation" synonymously with bimacular fusion and "monofixation" synonymously with absence of bimacular fusion but presence of peripheral fusion. The term "bifoveal fixation" used in reference to fixation disparity is accurate, since only the foveas determine the limits of tolerable inexactness by which the intersection of the visual axes may miss the fixation point.

said to have fixation disparity, but of a pathologic amount. Fusion disparity as a descriptive term has certain advantages." There are two obvious dissimilarities between fixation disparity* and the monofixation syndrome (Jampolsky's fusion disparity). The quantity of deviation does not exceed 6 to 10 minutes of arc in fixation disparity but may be as large as 8 prism diopters in the monofixation syndrome. In fixation disparity, both macular areas simultaneously function, whereas in the monofixation syndrome one or the other macula does not function during binocular vision.

It was apparent from the time I first became interested in the large group of patients having the monofixation syndrome that it would be impossible to accurately name this condition in accordance with the established semantic code in common usage for ocular motility and binocular vision. Appraised according to one respect, the patient was heterotropic, but in another respect he was heterophoric. Any term selected to identify these patients was arbitrary. In 1961 I applied the name monofixational phoria[44] to those patients with a deviation that was greater by alternate cover than by cover-uncover, claiming the deviation was made partially latent by peripheral fusion while the image projected onto the deviated macular area was suppressed. At the time, I was interested only in the small-angle deviations and was unaware that many patients without a deviation had the identical sensory abnormality of suppression of one macula. Jampolsky's concept of NRC peripheral fusion acquired by the normal stretched-out peripheral Panum's space was accepted, and the NRC seemed confirmed by the binocular perimetric finding performed during dissociated conditions. The other significant facts about the monofixation syndrome added by this report were:

1. Anisometropia, in addition to strabismus, was established as a cause.

2. In some patients neither strabismus nor anisometropia was present and these patients were defined as having primary monofixational phoria (those with strabismus and anisometropia having secondary monofixational phoria).

3. Stereoacuity was first related to the nature of fixation present, poor in monofixation and good in bifixation.

4. The facultative absolute scotoma was revealed by binocular perimetry.

*Fixation disparity is also known in the literature as retinal slip[3] (1928), cortical slip,[52] and fusional disparity[30] (1952), but none of these has been in serious contention as an acceptable synonym since the common usage of fixation disparity is so well established.

In 1962,[45] a statistical report supported the concepts established in the preceding year, and Albert[2] added more statistics to the literature. In 1964 Chamberlain and Caldwell[13] confirmed monofixational phoria to be a significant entity in ocular motility and endorsed the term as precise and descriptive. In the same year, I expressed misgivings about the original concept I held concerning NRC in these patients[40] because the binocular visuscope test and the image transfer test (better described in 1966[45]) indicated the presence of abnormal retinal correspondence (ARC). The Bagolini striated lens test provided the same finding.

At the First International Symposium on Strabismus in 1966, Lang[27] criticized monofixational phoria as a name for small angle strabismus since there is a manifest tropia* and the term was not compatible with the finding of anomalous correspondence. The small angle tropia to which he was referring was the well-known syndrome consisting of unilateral strabismus of less than 5 degrees, harmonious anomalous correspondence with partial stereopsis, and usually slight amblyopia in the non-fixating eye. Undoubtedly, this is the syndrome I called monofixational phoria and Lang admits calling strabismus spurius for several years. He now proposes that the full name of this syndrome be microtropia unilateralis anomalo-fusionalis, but suggests it be referred to ordinarily as microtropia or microstrabismus. Apparently, his choice was microtropia because thereafter he used this term when referring to the syndrome. Lang's criticism of monofixational phoria as a name for this syndrome is warranted on the basis that a shift in tropia is elicited by cover-uncover, but I am not convinced yet, although many sensory tests diagnose ARC, that actually ARC represents the true sensory status of these patients. For reasons that will be developed in this paper, there is still a good possibility that Jampolsky's original concept of NRC peripheral fusion is obtained by the stretched-out peripheral Panum's fusional space without an adaptation in retinal correspondence. Actually the ARC findings recorded in these patients are spurious. Lang has had considerable experience with the primary type of microtropia – patients who have never squinted. Apparently he considers the secondary type of microtropia secondary only to reduction by treatment of an angle of heterotropia larger than 5 degrees. He does not classify microtropia associated with anisometropia as secondary, as I did. He reports that, of 103 cases of primary microtropia in 32 children and 71 adults, only one was a

*Burian's[9] definition of heterophoria is "a deviation of the eyes kept latent by fusion; heterotropia is a patent (manifest) deviation of the eyes in the absence of fusion."

primary divergent microtropia. Sixty-five patients had central fixation in each eye by visuscopy, but 39 had eccentric fixation. He concludes that microtropia remains constant during life and conjectures that primary microtropia may deteriorate into a larger angle esotropia due to accommodative convergence or for other reasons. He also regrets that there is no way to know whether the microtropia resulting from reduction of larger angle heterotropia was originally a primary microtropia. In a second paper[27] he found that 19 per cent of 653 convergent strabismic patients have primary microtropia and 18.5 per cent have secondary microtropia. He emphasized that the increased familial incidence shows that a hereditary factor is involved in microstrabismus.

In 1967 another aspect was added to the monofixating syndrome. Helveston and von Noorden[17] described 20 amblyopic patients with a suppression scotoma whose non-preferred eye did not make a fixation movement upon covering the fixating eye. Despite the absence of proof by the cover test, a manifest deviation with ARC was presumed to be due to the visuscope target projecting onto the same retinal point in the amblyopic eye for both the monocular and binocular (bifoveal correspondence) tests. Since this retinal point was just adjacent to the macular border, the strabismic angle was ultra small – hence, they named the condition microtropia. Anisometropia was prevalent among their cases.

I can confirm the same findings in many patients with the monofixation syndrome whose poor-sighted eye either has not responded to therapy for amblyopia or has never been treated; it occurs either as a primary case or secondary to strabismus, anisometropia (or the two combined), or a macular lesion. I have referred to these cases as monofixational orthophoria since there is no detectable shift in either eye by the cover test. I agree with Helveston and von Noorden that actually there would be a discernible shift were it not for the slight eccentric fixation in the amblyopic eye; therefore, the patient is not orthophoric. However, in my opinion the term microtropia (Helveston and von Noorden) is not justifiably confined only to those patients whose heterotropia is inferred from visuscope findings. Nor should the term microstrabismus (Lang) be restricted to those patients whose heterotropia is obvious by cover tests, since microtropia and microstrabismus are synonymous. Moreover, it is contradictory for Helveston and von Noorden to use microtropia as they did, when Lang had previously suggested this term for another condition. In fact, the semantic structure that evolved as a result of attempts by many to

label various categories of patients that constitute the monofixation syndrome has become a monstrosity. Surely it will topple and such terms as retinal slip, fixation disparity, esophoria with fixation disparity, fixational disparity, flicker cases, subnormal binocular vision, convergent fixation disparity, pathological fixation disparity, monofixational phoria, fusion disparity, strabismus spurius, microtropia unilateralis anomalo-fusionalis, microtropia, and microstrabismus will vanish from ordinary usage.

There are three principal reasons for the past difficulties encountered in naming this syndrome: (1) an element of both phoria and tropia are present and whichever feature the author chooses to emphasize determines the selection; (2) fixation disparity, as a name for a specific physiologic process in binocular single vision, was quite naturally plagiarized, since the condition under discussion seemed to be a pathologic extension of the same process; and (3) the names selected revealed the lack of a total concept of the syndrome. As the syndrome was gradually put together in piecemeal fashion, the lack of organization in naming each of the facets is now very apparent.

Essentially the patients with this syndrome have a form of binocular single vision in which their inability to bifixate is proved by a demonstrable scotoma in the visual field of the non-fixating eye during binocular vision. Associated with this essential monofixating feature are other features, some always present and others sometimes present and sometimes absent. Always associated with the monofixation is fusion with vergence amplitudes and stereopsis. The variable features associated with this syndrome are a strabismus history, anisometropia, a unilateral macular lesion, amblyopia, eccentric fixation, orthophoria, phoria, small tropia, and possibly a deviation that is larger by alternate cover than by cover-uncover. The name that best fits all these features is simply "the monofixation syndrome."

## METHODS OF STUDY

The only criteria determining selection in this study were that a scotoma be proved to be within the binocular visual field, peripheral fusion with a fusional vergence be present, and there be at least 3000 seconds of arc of stereoacuity. Each patient included in this study is listed in Table 1 in the numerical order in which he appeared. The visual acuity examination was performed by use of entire lines of letters; visuscopy checked the quality of fixation.

Alignment was investigated by means of the cover test at both 20

TABLE 1

| Case number | Type of monofixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by | | | | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 1 | Acq ET | surg | 2 | 15 | 20/20 | foveal | +1.50+0.25×180 | ETcc =4 | 12′ | 14/13 | 1/1 | either eye | either eye | + | either eye | ARC | 120 |
| | | gl's | | | 20/20 | foveal | +0.25 | ET′cc =4 | 13″ | | | | | | | | 100 |
| 2 | Acq ET | surg | 4 | 15 | 20/20 | foveal | +3.50 | Ecc =10 | 10′ | 12/8 | 20/16 | either eye | either eye | − | LE | NRC | 0 |
| | | gl's | | | 20/20 | foveal | +4.50 | E′cc =18 | 13″ | | | | | | | | 200 |
| 3 | Primary | none | 16* | 16 | 20/20 | foveal | +0.25×115 | ETsc =2–4 | 10′ | 16/13 | 8/7 | either eye | either eye | + | LE | ARC | 120 |
| | | | | | 20/20 | foveal | +0.25+0.25×85 | ET′sc =8 | 13″ | | | | | | | | 67 |
| 4 | Aniso | gl's | 7* | 14 | 20/15 | foveal | +2.00 | Ortho cc | 8′ | 25/20 | 4/2 | LE | LE | + | LE | NRC | 120 |
| | | occl | | | 20/40 | foveal | +3.50+1.00×90 | Ortho′cc | 13″ | | | | | | | | 100 |
| 5 | Acq ET | bif | 3 | 12 | 20/20 | foveal | +0.25+2.00×80 | ETcc =1 | 9′ | 10/3 | 10/0 | either eye | either eye | + | RE | ARC | 0 |
| | | occl | | | 20/20 | foveal | +0.25+0.75×90 | ET′cc =3 | 13″ | | | | | | | | 400 |
| | | orth | | | | | | | | | | | | | | | |
| 6 | Acq XT | surg | 1 | 14 | 20/25 | foveal | +3.50+1.00×80 | Ortho cc | 10′ | 22/18 | 8/5 | either eye | either eye | − | either eye | NRC | 180 |
| | | gl's | | | 20/25 | foveal | +3.25+1.00×90 | Ortho′cc | 13″ | | | | | | | | 67 |
| 7 | Acq ET | miot | 1 1/2 | 13 | 20/20 | foveal | +0.75+0.50×180 | ETcc =1 | 8′ | 16/13 | 12/10 | LE | LE | + | LE | ARC | 0 |
| | | bif | | | 20/50 | perifoveal | +1.00 | ET′cc =22 | 13″ | | | | | | | | 400 |
| | | occl | | | | | | ET′cc(bif) =4 | | | | | | | | | |
| 8 | Acq ET | gl's | 2 1/2 | 14 | 20/15 | foveal | +5.00+1.25×90 | ETcc =4 | 10′ | 12/11 | 5/3 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | +5.75+1.75×85 | ET′cc =4 | 13″ | | | | | | | | 400 |
| 9 | Acq ET | gl's | 3 | 11 | 20/20 | foveal | +2.50+1.00×90 | Ortho cc | 11′ | 4/3 | 10/4 | either eye | none | − | RE | NRC | 0 |
| | | | | | 20/20 | foveal | +1.75+1.00×90 | E′cc =2 | 13″ | | | | | | | | 100 |
| 10 | Unilat | none | 8 1/2* | 12 | 20/20 | foveal | −0.50 | Ortho sc | 2′ | 12/10 | 6/5 | LE | LE | + | LE | ? | 0 |
| | Mac Les | | | | 2/200 | paramac | −0.50 | Ortho′sc | 6″ | | | | | | | | 100 |
| 11 | Primary | none | 10* | 10 | 20/25 | foveal | +1.50 | ETsc =6 | 11′ | 22/16 | 10/8 | LE | RE | + | RE | ARC | 120 |
| | | | | | 20/12.5 | foveal | +1.50 | ET′sc =4 | 13″ | | | | | | | | 67 |
| 12 | Aniso | gl's | 7* | 10 | 20/20 | foveal | +0.75+0.25×130 | Xcc =8 | 10′ | 14/12 | 17/6 | LE | LE | + | LE | NRC | 120 |
| | | | | | 20/25 | foveal | +2.75+1.50×80 | E′cc =8 | 13″ | | | | | | | | 100 |
| 13 | Acq ET | gl's | 7 | 10 | 20/20 | foveal | +6.75 | ETcc =4 | 9′ | 16/6 | 9/8 | LE | LE | + | LE | ARC | 0 |
| | and | occl | | | 20/50 | perifoveal | +6.25+2.00×90 | ET′cc =4 | 13″ | | | | | | | | 200 |
| | Aniso | | | | | | | | | | | | | | | | |
| 14 | Acq ET | bif | 2 | 10 | 20/20 | foveal | −0.25 | ETcc =6–12 | 10′ | 28/20 | 12/6 | LE | LE | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | −0.25 | ET′cc =42 | 13″ | | | | | | | | 1000 |
| | | | | | | | | ET′cc(bif) =8–20 | | | | | | | | | |

TABLE 1 (CONT.)

| Case number | Type of monofixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by | | | | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 15 | Acq ET | bif | 2 1/2 | 15 | 20/12.5 | foveal | +0.75 | ETcc = 6–12 | 12′ | 15/5 | 12/10 | LE | LE | + | LE | ARC | 0 |
| | | occl | | | 20/25 | foveal | +0.75 | ET′cc = 20 | 13″ | | | | | | | | 1000 |
| | | orth | | | | | | ET′cc(bif)6–10 | | | | | | | | | |
| 16 | Acq ET | bif | 2 1/2 | 11 | 20/40 | foveal | −3.00 | Ortho cc | 9′ | 15/13 | 8/4 | RE | RE | + | RE | ARC | 0 |
| | | occl | | | 20/25 | foveal | −3.50 | ET′cc = 1 | 13″ | | | | | | | | 200 |
| | | surg | | | | | | | | | | | | | | | |
| 17 | Acq ET | bif | 1 1/2 | 12 | 20/20 | foveal | +4.25+3.50×80 | Ortho cc | 10′ | 12/11 | 11/10 | either eye | either eye | − | RE | ARC | 0 |
| | | occl | | | 20/20 | foveal | +5.50+3.25×85 | ET′cc = 8–20 | 13″ | | | | | | | | 200 |
| | | surg | | | | | | | | | | | | | | | |
| 18 | Acq ET | gl's | 1/2 | 11 | 20/20 | foveal | +2.75+1.50×10 | Ortho cc | 15′ | 11/9 | 11/9 | either eye | none | − | LE | NRC | 180 |
| | | miot | | | 20/25 | foveal | +3.50+1.75×170 | Ortho′cc | 13″ | | | | | | | | 200 |
| | | surg | | | | | | | | | | | | | | | |
| 19 | Acq XT and Aniso | gl's | 3 1/2 | 8 | 20/20 | foveal | +5.00+0.50×90 | Ortho cc | 11′ | 24/12 | 19/12 | either eye | either eye | − | RE | NRC | 120 |
| | | surg | | | 20/20 | foveal | +3.00+0.50×90 | Ortho′cc | 13″ | | | | | | | | 67 |
| 20 | Acq ET | bif | 2 | 11 | 20/20 | foveal | −0.75+0.50×20 | ETcc = 6–12 | 8′ | 19/18 | 16/16 | either eye | either eye | + | LE | ARC | 120 |
| | | | | | 20/30 | foveal | −0.25+0.75×160 | ET′cc = 20 | 13″ | | | | | | | | 200 |
| | | | | | | | | ET′cc(bif)6–10 | | | | | | | | | |
| 21 | Primary | gl's | 7* | 14 | 20/25 | foveal | −2.25+2.00×20 | Ortho cc | 9′ | 20/10 | 3/1 | either eye | either eye | − | RE | NRC | 0 |
| | | | | | 20/20 | foveal | −2.25+1.50×5 | Ortho′cc | 13″ | | | | | | | | 400 |
| 22 | Acq ET | gl's | 5 | 8 | 20/20 | foveal | +3.00+0.50×90 | Ortho cc | 9′ | 21/15 | 11/6 | either eye | LE | + | LE | NRC | 180 |
| | | occl | | | 20/25 | foveal | +4.50 | Ortho′cc | 13″ | | | | | | | | 100 |
| | | surg | | | | | | | | | | | | | | | |
| 23 | Cong ET | surg | | 9 | 20/20 | foveal | +0.25+0.25×30 | Ortho cc | 8′ | 22/16 | 5/4 | either eye | either eye | − | LE | NRC | 0 |
| | | gl's | | | 20/20 | foveal | −1.00+0.25×150 | Ortho′cc | 13″ | | | | | | | | 3000 |
| 24 | Primary | occl | 6* | 10 | 20/50 | foveal | −0.50 | ETsc = 4–12 | 8′ | 13/11 | 12/10 | RE | RE | + | RE | ARC | 0 |
| | | orth | | | 20/20 | foveal | −0.50 | ET′sc = 4–6 | 13″ | | | | | | | | 400 |
| 25 | Acq ET | gl's | 2 | 15 | 20/20 | foveal | +5.75+0.50×40 | ETcc = 2 | 13′ | 12/11 | 11/10 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/25 | foveal | +6.75 | ET′cc = 2 | 13″ | | | | | | | | 200 |
| 26 | Primary | gl's | 7* | 15 | 20/20 | foveal | −2.50 | ETcc = 4 | 10′ | 28/26 | 10/8 | either eye | either eye | + | LE | ARC | 240 |
| | | occl | | | 20/30 | foveal | −2.50 | ET′cc = 4 | 13″ | | | | | | | | 400 |
| 27 | Acq ET | occl | 2 | 8 | 20/20 | foveal | +1.00 | ETcc = 6–10 | 12′ | 15/10 | 6/5 | either eye | either eye | + | LE | ARC | 0 |
| | | surg | | | 20/20 | foveal | +0.75 | ET′cc = 6–12 | 13″ | | | | | | | | 100 |

TABLE 1 (CONT.)

| Case number | Type of mono-fixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by | | | | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 28 | Acq ET | gl's | 2 1/2 | 14 | 20/20 | foveal | +3.00+1.00×90 | ETcc =2 | 15′ | 24/20 | 13/12 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | +3.50+2.00×65 | ET′cc =2 | 13″ | | | | | | | | 200 |
| 29 | Primary | gl's | 6* | 11 | 20/20 | foveal | −6.50+0.50×180 | Ortho cc | 9′ | 16/16 | 6/3 | either eye | either eye | − | RE | NRC | 0 |
| | | | | | 20/20 | foveal | −6.50 | Ortho′cc | 13″ | | | | | | | | 400 |
| 30 | Acq ET | gl's | 4 | 11 | 20/15 | foveal | +3.25 | ETcc =4 | 9′ | 18/17 | 9/8 | LE | LE | + | LE | ARC | 0 |
| | | occl | | | 20/70 | perifoveal | +4.00+0.50×180 | ET′cc =15<br>ET′cc(bif) =4 | 13″ | | | | | | | | 400 |
| 31 | Acq ET | gl's | 1/2 | 14 | 20/20 | foveal | −0.75+3.00×10 | Ortho cc | 10′ | 18/10 | 16/8 | either eye | either eye | − | LE | ARC | 240 |
| | | occl | | | 20/30 | foveal | −1.00+2.25×180 | ET′cc =2 | 13″ | | | | | | | | 200 |
| 32 | Primary | none | 12* | 12 | 20/12.5 | foveal | plano | ETsc =2–4 | 8′ | 20/18 | 12/10 | LE | either eye | + | LE | ARC | 0 |
| | | | | | 20/25 | foveal | plano | ET′sc =2–6 | 13″ | | | | | | | | 100 |
| 33 | Acq ET | gl's | 4 | 9 | 20/20 | foveal | +2.00+0.50×90 | ETcc =2 | 11′ | 30/20 | 14/12 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | +2.00 | ET′cc =4 | 13″ | | | | | | | | 1000 |
| | | surg | | | | | | | | | | | | | | | |
| 34 | Primary | gl's | 12* | 13 | 20/25 | foveal | −0.25+0.50×180 | Ortho cc | 18′ | 25/19 | 11/9 | either eye | RE | + | RE | NRC | 180 |
| | | | | | 20/20 | foveal | −1.00+0.25×5 | Ortho′cc | 13″ | | | | | | | | 200 |
| 35 | Aniso | gl's | 7* | 7 | 20/20 | foveal | −0.50+0.75×40 | Ortho cc | 11′ | 6/4 | 14/12 | LE | LE | + | LE | NRC | 120 |
| | | | | | 20/30 | foveal | −2.50+2.75×120 | Ortho′cc | 13″ | | | | | | | | 100 |
| 36 | Acq XT | surg | 2 | 12 | 20/20 | foveal | +0.50 | Xsc =4 | 10′ | 24/20 | 16/14 | either eye | either eye | − | RE | NRC | 0 |
| | | | | | 20/20 | foveal | +0.50 | Ortho′sc | 13″ | | | | | | | | 200 |
| 37 | Acq ET | gl's | 1 | 14 | 20/20 | foveal | +2.50+0.50×90 | Ortho cc | 12′ | 22/19 | 16/14 | either eye | either eye | − | RE | NRC | 0 |
| | | | | | 20/20 | foveal | +3.00+0.75×110 | Ortho′cc | 13″ | | | | | | | | 200 |
| 38 | Acq ET | gl's | 1 1/2 | 10 | 20/25 | foveal | +2.50 | Ortho cc | 8′ | 14/10 | 10/8 | either eye | none | + | RE | NRC | 0 |
| | | surg | | | 20/12.5 | foveal | +2.50 | Ortho′cc | 13″ | | | | | | | | 1000 |
| 39 | Acq ET and aniso | gl's | 5 | 11 | 20/20 | foveal | +4.75+0.50×10 | Ortho cc | 9′ | 30/30 | 4/3 | LE | LE | + | LE | NRC | 0 |
| | | occl | | | 20/30 | foveal | +6.00+1.25×170 | Ortho′cc | 13″ | | | | | | | | 67 |
| 40 | Cong ET | surg | | 13 | 20/30 | foveal | +0.25 | Ortho sc | 10′ | 10/10 | 8/4 | none | RE | + | RE | NRC | 0 |
| | | occl | | | 20/12.5 | foveal | +0.25 | Ortho′sc | 13″ | | | | | | | | 3000 |
| 41 | Acq ET | gl's | 3 1/2 | 13 | 20/12.5 | foveal | +3.25+1.50×105 | ETcc =4 | 8′ | 25/22 | 9/8 | either eye | either eye | + | RE | ARC | 120 |
| | | occl | | | 20/12.5 | foveal | +3.50+0.75×75 | ET′cc =6 | 13″ | | | | | | | | 67 |
| | | surg | | | | | | | | | | | | | | | |

TABLE 1 (CONT.)

| Case number | Type of mono-fixation | Treat-ment received | Age of onset or dis-covery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, dis-tance and near | Fusional conver-gence ampli-tude | Fusional diver-gence ampli-tude | Scotoma elicited by | | | | Retinal corre-spondence striated lenses | Stereo-acuity, distance and near (in sec-onds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 42 | Acq ET and aniso | bif occl surg | 2 1/2 | 13 | 20/20<br>20/80 | foveal<br>perimac | +4.50<br>+6.50 | Ortho cc<br>Ortho'cc | 7′<br>13″ | 14/4 | 12/2 | LE | LE | + | LE | ? | 240<br>67 |
| 43 | Primary | none | 9* | 16 | 20/20<br>20/40 | foveal<br>foveal | +0.50<br>+0.50+0.75×80 | Ortho sc<br>Ortho'sc | 10′<br>13″ | 19/18 | 10/8 | LE | LE | + | LE | NRC | 180<br>100 |
| 44 | Acq ET | gl's | 1 1/2 | 10 | 20/20<br>20/12.5 | foveal<br>foveal | +7.25+2.25×85<br>+7.00+2.00×85 | XTcc =2<br>XT'cc =2 | 8′<br>13″ | 20/18 | 5/5 | either eye | RE | + | RE | ARC | 240<br>400 |
| 45 | Primary | none | 8* | 12 | 20/12.5<br>20/25 | foveal<br>foveal | +1.00<br>+1.00 | ETsc =4–12<br>ET'sc =8–18 | 10′<br>13″ | 26/22 | 6/0 | LE | LE | + | LE | ARC | 0<br>200 |
| 46 | Acq ET and aniso | gl's occl | 4 | 12 | 20/20<br>20/30 | foveal<br>foveal | +5.50+2.50×100<br>+7.50+2.50×115 | Ortho cc<br>ET'cc =2 | 14′<br>13″ | 13/11 | 12/11 | either eye | LE | + | LE | ARC | 240<br>100 |
| 47 | Acq ET | occl surg | 5 1/2 | 16 | 20/20<br>20/10 | foveal<br>foveal | +0.25×180<br>+0.25×10 | ETsc =4<br>ET'sc =6 | 9′<br>13″ | 16/14 | 15/14 | either eye | either eye | + | RE | ARC | 0<br>200 |
| 48 | Acq ET | gl's | 4 1/2 | 13 | 20/15<br>20/15 | foveal<br>foveal | −1.00+0.25×10<br>−0.75 | Ecc =8<br>ET'cc =2 | 12′<br>13″ | 22/19 | 12/8 | either eye | either eye | − | LE | ARC | 0<br>200 |
| 49 | Acq ET | bif orth surg | 1 1/2 | 13 | 20/20<br>20/20 | foveal<br>foveal | +2.25+1.50×95<br>+3.00+0.75×105 | Ecc =4<br>ET'cc =2–10 | 10′<br>13″ | 20/18 | 15/14 | either eye | either eye | − | RE | ARC | 0<br>200 |
| 50 | Acq XT | surg | 1 | 12 | 20/30<br>20/25 | foveal<br>foveal | −0.75<br>−0.75 | XTsc =4<br>LHT'sc =2 | 11′<br>13″ | 27/24 | 6/5 | either eye | either eye | + | RE | ARC | 0<br>100 |
| 51 | Acq ET | gl's surg | 3 | 8 | 20/25<br>20/20 | foveal<br>foveal | +4.00+0.50×90<br>+4.00 | ETcc =2<br>ET'cc =6 | 8′<br>13″ | 17/16 | 8/6 | either eye | either eye | + | RE | ARC | 0<br>200 |
| 52 | Primary | gl's occl | 7* | 8 | 20/20<br>20/20 | foveal<br>foveal | +1.00+0.75×90<br>+1.00+0.75×90 | ETcc =4<br>ET'cc =4 | 10′<br>13″ | 26/21 | 7/6 | either eye | either eye | + | LE | ARC | 120<br>67 |
| 53 | Acq ET | gl's occl | 7 | 11 | 20/30<br>20/25 | foveal<br>foveal | +3.25<br>+2.50 | ETcc =6<br>ET'cc +6–12 | 10′<br>13″ | 28/26 | 19/14 | either eye | either eye | + | RE | ARC | 0<br>400 |
| 54 | Acq XT | occl gl's surg | 2 | 11 | 20/20<br>20/20 | foveal<br>foveal | −0.50+0.50×150<br>−0.50+1.00×175 | ETcc =4<br>ET'cc =8 | 9′<br>13″ | 20/10 | 10/8 | either eye | either eye | + | RE | ARC | 0<br>1000 |
| 55 | Acq ET | gl's | 1/2 | 12 | 20/20<br>20/20 | foveal<br>foveal | +3.00+0.50×80<br>+2.75+0.50×80 | Ortho cc<br>ET'cc =2–8 | 15′<br>13″ | 16/16 | 8/8 | either eye | either eye | − | alternates | ARC | 240<br>100 |

TABLE 1 (CONT.)

| Case number | Type of monofixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by | | | | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 56 | Primary | gl's | 8* | 11 | 20/25 | foveal | −3.00 | ETcc =2 | 8′ | 20/18 | 12/10 | either eye | either eye | − | RE | ARC | 0 |
| | | | | | 20/25 | foveal | −2.00 | ET′cc =2 | 13″ | | | | | | | | 100 |
| 57 | Aniso | gl's | 7* | 9 | 20/15 | foveal | +0.50+0.50×90 | ETcc =4 | 7′ | 20/16 | 10/8 | LE | LE | + | LE | ARC | 240 |
| | | | | | 20/30 | foveal | +2.75+1.00×90 | ET′cc =4 | 13″ | | | | | | | | 200 |
| 58 | Acq ET | gl's | 6 | 11 | 20/30 | foveal | +1.75+1.00×90 | ETcc =2 | 9′ | 20/18 | 14/12 | either eye | either eye | + | RE | ARC | 0 |
| | | occl | | | 20/20 | foveal | +1.50+1.00×90 | ET′cc =2–6 | 13″ | | | | | | | | 1000 |
| | | surg | | | | | | | | | | | | | | | |
| 59 | Primary | none | 10* | 10 | 20/30 | foveal | −0.75+1.00×20 | ETcc =8–14 | 10′ | 22/21 | 10/8 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/20 | foveal | plano | ET′cc =6–16 | 13″ | | | | | | | | 200 |
| 60 | Acq ET | occl | 6 | 10 | 20/20 | foveal | +1.50 | Ortho sc | 10′ | 27/24 | 13/12 | RE | RE | − | RE | NRC | 0 |
| | | surg | | | 20/15 | foveal | +1.25 | Ortho′sc | 13″ | | | | | | | | 400 |
| 61 | Acq ET | bif | 1 | 10 | 20/20 | foveal | +2.00 | Ortho cc | 13′ | 20/19 | 10/9 | either eye | either eye | − | RE | ARC | 0 |
| | | occl | | | 20/20 | foveal | +2.00 | ET′cc =6–14 | 13″ | | | | | | | | 100 |
| | | surg | | | | | | | | | | | | | | | |
| 62 | Acq ET | gl's | 2 1/2 | 15 | 20/20 | foveal | +4.25+1.25×105 | Ortho cc | 10′ | 15/14 | 9/8 | either eye | either eye | − | RE | NRC | 120 |
| | and | occl | | | 20/20 | foveal | +3.50+3.25×75 | E′cc =10 | 13″ | | | | | | | | 200 |
| | aniso | | | | | | | | | | | | | | | | |
| 63 | Cong ET | surg | | 7 | 20/12.5 | foveal | plano | Ortho sc | 10′ | 17/15 | 8/7 | either eye | either eye | − | LE | NRC | 240 |
| | | | | | 20/12.5 | foveal | plano | Ortho′sc | 13″ | | | | | | | | 400 |
| 64 | Acq ET | gl's | 3 | 11 | 20/30 | foveal | +4.75+2.25×85 | Ortho cc | 10′ | 13/9 | 9/8 | RE | RE | + | RE | NRC | 0 |
| | | occl | | | 20/15 | foveal | +3.75+1.25×85 | Ortho′cc | 13″ | | | | | | | | 200 |
| 65 | Acq XT | surg | 1 | 10 | 20/12.5 | foveal | +1.00 | XTsc =8 | 11′ | 12/4 | 4/2 | LE | LE | + | LE | ARC | 0 |
| | | orth | | | 20/20 | foveal | +1.00 | XT′sc =6 | 13″ | | | | | | | | 1000 |
| 66 | Acq ET | gl's | 2 1/2 | 12 | 20/20 | foveal | +7.25+1.00×90 | ETcc =2 | 10′ | 21/19 | 8/7 | either eye | either eye | + | LE | ARC | 0 |
| | | orth | | | 20/25 | foveal | +7.50+1.25×80 | ET′cc =4 | 13″ | | | | | | | | 1000 |
| 67 | Acq ET | bif | 4 | 12 | 20/15 | foveal | +1.50 | ETcc =2–8 | 12′ | 10/4 | 17/12 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/15 | foveal | +1.50 | ET′cc =20 | 13″ | | | | | | | | 200 |
| | | | | | | | | ET′cc(bif) =48 | | | | | | | | | |
| 68 | Acq ET | surg | 2 | 10 | 20/20 | foveal | +0.50 | Ortho sc | 10′ | 25/21 | 9/5 | either eye | RE | − | RE | NRC | 0 |
| | | occl | | | 20/20 | foveal | +0.50 | Ortho′sc | 13″ | | | | | | | | 400 |
| 69 | Acq ET | bif | 2 | 11 | 20/20 | foveal | +5.25+1.75×80 | ETcc =2–14 | 9′ | 6/6 | 4/4 | LE | LE | + | LE | ARC | 0 |
| | | occl | | | 20/50 | perifoveal | +5.75+2.50×100 | ET′cc =25 | 13″ | | | | | | | | 400 |
| | | | | | | | | ET′cc(bif) =4 | | | | | | | | | |

TABLE 1 (CONT.)

| Case number | Type of monofixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by | | | | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | | | | Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | | |
| 70 | Primary | none | 11* | 11 | 20/20 | foveal | +1.25+0.50×10 | ETsc =2–6 | 8′ | 22/17 | 12/10 | either eye | none | + | LE | ARC | 0 |
| | | | | | 20/25 | foveal | +0.75+0.75×170 | ET′sc =2–6 | 13″ | | | | | | | | 200 |
| 71 | Acq ET | bif miot occl | 4 1/2 | 16 | 20/25 | foveal | −2.75 | ETcc =4 | 8′ | 27/24 | 17/14 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/15 | foveal | −2.00+0.50×90 | ET′cc =6 | 13″ | | | | | | | | 1000 |
| 72 | Primary | none | 8* | 14 | 20/20 | foveal | −0.50+0.50×15 | ETcc =2 | 13′ | 23/21 | 15/13 | LE | LE | + | LE | ARC | 0 |
| | | | | | 20/30 | foveal | −0.25+0.25×125 | ET′cc =2 | 13″ | | | | | | | | 400 |
| 73 | Acq ET | surg gl's occl | 1 1/2 | 10 | 20/12.5 | foveal | +1.75+1.25×90 | ETcc =4 | 10′ | 20/15 | 14/10 | LE | LE | + | LE | ARC | 0 |
| | | | | | 20/30 | foveal | +2.50+1.50×90 | ET′cc =6 | 13″ | | | | | | | | 100 |
| 74 | Acq ET | gl's surg | 6 | 8 | 20/20 | foveal | +3.00+0.50×80 | ETcc =1 | 9′ | 20/18 | 12/10 | either eye | either eye | − | LE | NRC | 0 |
| | | | | | 20/25 | foveal | +2.25+1.25×95 | Ortho′cc | 13″ | | | | | | | | 400 |
| 75 | Acq ET | gl's occl surg | 2 | 12 | 20/20 | foveal | +4.50+1.25×90 | ETcc =6;RHT =5 | 12′ | 16/11 | 16/8 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/20 | foveal | +3.75+1.00×80 | ET′cc =6;RHT′=5 | 13″ | | | | | | | | 100 |
| 76 | Cong ET | surg gl's occl | | 16 | 20/15 | foveal | +2.50 | Ortho cc | 7′ | 15/15 | 15/15 | either eye | either eye | − | alternates | NRC | 0 |
| | | | | | 20/20 | foveal | +2.50 | Ortho′cc | 13″ | | | | | | | | 100 |
| 77 | Acq ET | miot occl surg | 6 | 9 | 20/20 | foveal | +0.75 | ETcc =4 | 10′ | 29/21 | 15/13 | either eye | either eye | + | LE | ARC | 0 |
| | | | | | 20/30 | foveal | +0.75 | ET′cc =6 | 13″ | | | | | | | | 200 |
| 78 | Acq ET | gl's occl surg | 2 1/2 | 10 | 20/40 | foveal | +3.50+1.25×90 | ETcc =4–6 | 10′ | 17/14 | 10/8 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/20 | foveal | +3.00+1.25×90 | ET′cc =6–8 | 13″ | | | | | | | | 200 |
| 79 | Acq ET | gl's | 5 | 9 | 20/20 | foveal | +6.25+0.75×75 | Ortho cc | 14′ | 25/21 | 10/8 | RE | none | − | RE | NRC | 120 |
| | | | | | 20/20 | foveal | +6.25+1.25×95 | E′cc =4 | 13″ | | | | | | | | 67 |
| 80 | Acq ET | gl's | 4 | 16 | 20/30 | foveal | +3.50 | ETcc =4 | 10′ | 14/12 | 6/4 | either eye | either eye | + | RE | ARC | 0 |
| | | | | | 20/15 | foveal | +3.00 | ET′cc =3–12 | 13″ | | | | | | | | 400 |
| 81 | Acq ET | gl's occl surg | 1 | 9 | 20/15 | foveal | +7.00+1.50×90 | ETcc =4 | 8′ | 20/8 | 0 | LE | LE | + | LE | ARC | 0 |
| | | | | | 20/30 | foveal | +7.25+1.50×85 | ET′cc =4 | 13″ | | | | | | | | 3000 |
| 82 | Acq ET and aniso | gl's occl surg | 3 1/2 | 15 | 20/25 | foveal | +3.75+1.75×15 | ETcc =2 | 10′ | 22/17 | 17/15 | either eye | RE | + | RE | ARC | 0 |
| | | | | | 20/20 | foveal | +5.50 | ET′cc =2 | 13″ | | | | | | | | 1000 |
| 83 | Primary | occl | 6* | 14 | 20/40 | perifoveal | +0.37 | Ortho sc | 9′ | 12/11 | 12/10 | RE | RE | + | RE | NRC | 0 |
| | | | | | 20/12.5 | foveal | +0.37 | Ortho′sc | 13″ | | | | | | | | 200 |

TABLE 1 (CONT.)

| Case number | Type of monofixation | Treatment received | Age of onset or discovery | Present age | Visual acuity | Visuscopy | Refraction | Alignment, distance and near | Worth, distance and near | Fusional convergence amplitude | Fusional divergence amplitude | Scotoma elicited by: Binocular perimetry | Striated lenses | 4Δ B-O test | Polaroid vectograph | Retinal correspondence striated lenses | Stereoacuity, distance and near (in seconds of arc) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 84 | Acq ET | gl's | 4 | 10 | 20/20 | foveal | −0.25+1.75×25 | ETcc=6–14 | 10′ | 20/17 | 8/7 | either eye | either eye | + | LE | ARC | 120 |
| | | occl | | | 20/20 | foveal | +0.50+2.00×160 | ET′cc=4–8 | 13″ | | | | | | | | 100 |
| 85 | Aniso | gl's | 6* | 7 | 20/15 | foveal | +1.50 | Ortho cc | 11′ | 20/10 | 8/4 | LE | LE | + | LE | NRC | 120 |
| | | occl | | | 20/20 | foveal | +3.25 | Ortho′cc | 13″ | | | | | | | | 100 |
| 86 | Acq ET | gl's | 1 1/2 | 10 | 20/15 | foveal | +4.50+0.25×85 | ETcc=3 | 8′ | 16/9 | 9/8 | either eye | either eye | + | LE | ARC | 0 |
| | | surg | | | 20/15 | foveal | +4.25+0.50×85 | ET′cc=6 | 13″ | | | | | | | | 200 |
| 87 | Acq XT | surg | 1 | 18 | 20/20 | foveal | −1.50 | Ecc=2;LH=4 | 12′ | 36/30 | 8/6 | LE | LE | + | LE | NRC | 0 |
| | | gl's | | | 20/20 | foveal | −1.50 | E′cc=2;LH′=4 | 13″ | | | | | | | | 200 |
| 88 | Primary | occl | 6* | 7 | 20/20 | foveal | +1.25 | Ortho sc | 10′ | 18/15 | 10/8 | LE | LE | + | LE | NRC | 0 |
| | | | | | 20/30 | foveal | +1.25 | Ortho′sc | 13″ | | | | | | | | 1000 |
| 89 | Cong ET | surg | | 9 | 20/20 | foveal | +1.25 | Ortho sc | 10′ | 22/16 | 8/6 | either eye | either eye | − | RE | NRC | 120 |
| | | | | | 20/20 | foveal | +1.25 | Ortho′sc | 13″ | | | | | | | | 67 |
| 90 | Acq ET | bif | 2 | 11 | 20/30 | foveal | +0.75 | ETcc=4 | 9′ | 26/20 | 11/7 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | +1.50+0.25×90 | ET′cc=16 | 13″ | | | | | | | | 1000 |
| | | surg | | | | | | ET′cc(bif)=4 | | | | | | | | | |
| 91 | Acq ET | gl's | 2 1/2 | 9 | 20/20 | foveal | +2.00 | ETcc=3–7 | 10′ | 18/16 | 15/12 | LE | LE | + | LE | ARC | 0 |
| | | surg | | | 20/25 | foveal | +2.50 | ET′cc=6–14 | 13″ | | | | | | | | 200 |
| 92 | Aniso | gl's | 8 1/2* | 11 | 20/25 | foveal | +2.75 | Ortho cc | 10′ | 19/17 | 5/4 | RE | RE | + | RE | NRC | 0 |
| | | occl | | | 20/12.5 | foveal | +0.25 | Ortho′cc | 13″ | | | | | | | | 200 |
| 93 | Acq ET | bif | 3 | 13 | 20/50 | perifoveal | +3.50+2.25×95 | ETcc=2 | 8′ | 22/20 | 5/3 | RE | RE | + | RE | ARC | 0 |
| | | occl | | | 20/20 | foveal | +3.75+1.75×75 | ET′cc=4–10 | 13″ | | | | | | | | 400 |
| | | surg | | | | | | | | | | | | | | | |
| 94 | Acq ET | gl's | 4 | 10 | 20/20 | foveal | −0.25+2.00×100 | Ortho cc | 10′ | 42/40 | 11/6 | either eye | RE | − | RE | NRC | 0 |
| | | occl | | | 20/20 | foveal | +0.25+1.75×60 | Ortho′cc | 13″ | | | | | | | | 200 |
| | | surg | | | | | | | | | | | | | | | |
| 95 | Acq ET | gl's | 4 | 13 | 20/20 | foveal | +3.50 | ETcc=3–10 | 13′ | 29/28 | 12/9 | LE | none | + | LE | ARC | 0 |
| | | | | | 20/20 | foveal | +4.00 | ET′cc=3–16 | 13″ | | | | | | | | 1000 |
| 96 | Primary | none | 8* | 14 | 20/40 | foveal | +1.00 | Ortho sc | 10′ | 22/20 | 8/7 | RE | RE | + | RE | NRC | 120 |
| | | | | | 20/20 | foveal | +1.00 | E′sc=10 | 13″ | | | | | | | | 100 |
| 97 | Acq ET and aniso | gl's | 6 | 12 | 20/20 | foveal | +1.75+0.75×5 | ETcc=1 | 10′ | 17/14 | 3/2 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/30 | foveal | +3.50+0.50×85 | ET′cc=2 | 13″ | | | | | | | | 200 |
| 98 | Primary | none | 10* | 10 | 20/15 | foveal | −0.25 | Xsc=2 | 9′ | 28/25 | 12/10 | either eye | LE | − | LE | NRC | 0 |
| | | | | | 20/15 | foveal | −0.25 | X′sc=4 | 13″ | | | | | | | | 200 |
| 99 | Cong ET | surg | | 15 | 20/15 | foveal | +0.75 | ETsc=2–5 | 8′ | 20/17 | 6/4 | either eye | either eye | − | alternates | ARC | 120 |
| | | | | | 20/15 | foveal | +0.50 | ET′cc=4–6 | 13″ | | | | | | | | 100 |
| 100 | Acq ET | gl's | 1 1/2 | 12 | 20/20 | foveal | +0.25+0.50×80 | ETcc=6–10 | 10′ | 18/12 | 12/11 | either eye | either eye | + | LE | ARC | 0 |
| | | occl | | | 20/25 | foveal | +0.75+0.75×100 | ET′cc=6–12 | 13″ | | | | | | | | 200 |
| | | surg | | | | | | | | | | | | | | | |

*Age of discovery.

feet and 13 inches. Two adaptations of the cover test were used: first, cover-uncover performed on each eye; second, prism and alternate cover. If a deviation was disclosed by the cover-uncover test, it was measured by the simultaneous prism and cover test; i.e., the prism power was placed before the deviating eye as the fixating eye was covered, neutralizing all movement. If there was a difference in the deviations disclosed by these two cover tests, both values are listed in Table 1, the first figure being the simultaneous prism and cover test finding. If both the simultaneous prism and cover test and prism and alternate cover test findings were identical, only one value is listed. The accommodation was controlled during the cover tests by compensating for a significant refractive error with lenses and using small print as fixation targets for both distance and near.

A difference of greater than 1.50 diopters between the two eyes in spherical equivalent or astigmatism was the criterion used to designate anisometropia.

The sensory status of each patient was investigated by a battery of tests designed to demonstrate fusion, search for a scotoma, evaluate retinal correspondence, and quantitate stereopsis.

The horizontal fusional vergence amplitude was determined while the patient read small Snellen letters at 20 feet, projected on a 2.5-degree screen in a well-illuminated room. Surrounding the screen were multiple small pictures, and 4 degrees below the screen center was a bright blinking light. These surroundings to the fixation target offered ample opportunity for diplopia recognition when the fusional vergence amplitude was exceeded. The base-in and then the base-out power was increased, using a rotary prism, until diplopia or blurred vision was reported. Both the break and restoration points were measured. The amplitudes recorded in Table 1 are adjusted according to the prism and alternate cover finding, representing actual fusional vergence amplitudes.

Five different methods were used to identify the presence of the monocular scotoma in the binocular visual field. The first method used was the Worth four lights at both 20 feet and 13 inches. The distance test projected onto a 1.25-degree retinal area and the near test onto a 6-degree area. Those patients who were unable to fuse the lights at 20 feet or 13 inches slowly approached them until they could; in such instances, this distance was then recorded.

The second method was to study the central field of each eye by plotting the binocular field at a distance of 1 meter in a darkened room. A $1 \cdot m^2$ white vision screen with a central 5-mm fixation "0"

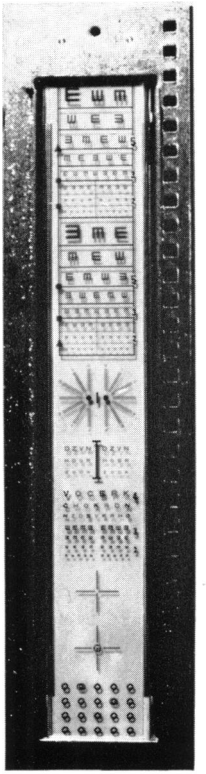
surrounded by 5-degree concentric circle markings was illuminated by diffuse red light. A beam of green light from a mounted movable projector projected a sharply focused 1-mm circular test target on the screen. A red filter was placed before one eye and a green filter before the other. As the eye behind the red filter fixated the stationary central target, the other eye behind the green filter saw only the movable green test target. The patient's hands guided the projector as the green target was steered about the screen while attempting to superimpose it upon the fixation target. A bifixating patient directly superimposed the target. A monofixating patient was unable to accomplish this task because the test target disappeared within the scotoma of the non-fixating eye as it approached the fixation target and reappeared on the other side of the target as it emerged from the scotoma. The disappearance and reappearance of the test target allowed the examiner to plot size and shape of the scotoma.

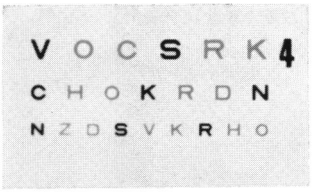
The third method used to diagnose the presence of a scotoma involved the four-diopter base-out prism test described by Irvine.[19] While the patient read letters at 20 feet, a four-diopter base-out prism was slipped before first one eye and then the other; the prism-covered eye was watched closely to determine if movement occurred. Absence of movement was interpreted as proof of a scotoma in the visual field of that eye, granted that the other eye moved in response to the prism placed before it. However, if neither eye moved in response to the prism placed in sequence before each, the test result was scored as negative.

Bagolini striated lenses were utilized in the fourth method for investigation of the scotoma. The patient was taught to recognize his own scotoma and report on it while viewing a small hand-held muscle light 15 inches away in a normally illuminated room. The lenses were positioned so that the streak seen by the right eye was at 135 degrees and that seen by the left eye was at 45 degrees. A scotoma was seen by most patients as a gap around the light in the streak seen by the non-fixating eye. After observing the scotoma by this technique in the visual field of one eye, the patient was encouraged to switch fixation to the other eye to observe whether the scotoma was transferred to the visual field of the other eye.

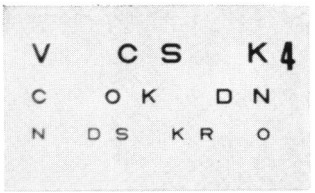
The fifth method to investigate the scotoma made use of the A-O Vectographic Project-O-Chart Slide* (Figure 1) in conjunction with a non-depolarizing aluminized screen.† Each character on the slide

*American Optical Company Catalog No. 11245 Adult Slide.
†American Optical Company.

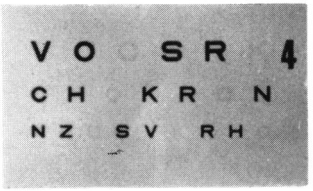





Seen by both eyes




Seen by right eye




Seen by left eye

FIGURE 1

had self-contained light polarizations, some being polarized at 90 degrees to others. Viewed through analyzers, some images were made visible to one eye and invisible to the other, whereas some characters were common to both eyes. This method provided a test environment closely approximating the normal binocular situation. The bifixator read the entire 20/40 line without hesitation although two letters were seen only by the right eye, two others only by the left eye, and the remaining two letters by both eyes. The monofixator deleted the two letters that were imaged only in the non-fixating eye. Occasionally, a monofixator who rapidly alternated fixation from one eye to the other would read all six letters, but usually he would comment that as two letters disappeared two others appeared.

Retinal correspondence was assessed with Bagolini striated lenses. A person with straight eyes and NRC saw the streaks intersecting at

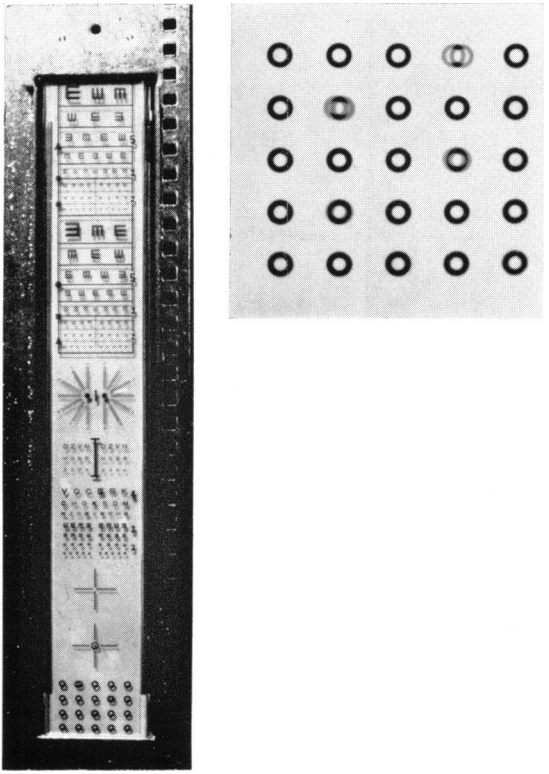


FIGURE 2
The range of stereoacuity from top to bottom is 240, 180, 120, 60, and 30 seconds of arc.

the light forming an X. A patient with deviated eyes and NRC saw two lights with one streak passing through each light. A patient with deviated eyes and ARC saw one light and both streaks passing through the light. If no deviation was elicited by cover-uncover in a patient having eccentric fixation in the amblyopic eye established by visuscopy and who claimed the streaks passed through the light, unknown retinal correspondence was recorded.

Stereopsis was graded according to the least horizontal retinal image disparity that evoked this perception. The determination of stereoacuity was measured in seconds of arc of image disparity. Polaroid vectographs produced the image disparity, and the patient viewed them through polaroid analyzers in normal room illumination. The analyzers were fitted over the patient's spectacles, if glasses were

worn. The stereoacuity was determined at both 20 feet and 16 inches. The distant vectographs were projected onto a non-depolarizing aluminized screen* using the A-O Vectographic Project-O-Chart Slide† (Figure 2). This slide provides five different gradations of stereoacuity, ranging from 30 to 240 seconds of arc. Two vectographic plates were used to determine the stereoacuity at 16 inches. The Wirt Stereotest‡ provided 12 different gradations of stereoacuity, ranging from 14 to 1000 seconds of arc, and the Stereo Fly Test‡ produced approximately 3000 seconds of arc of image disparity.

## RESULTS

Of the 100 patients in this study, 53 were male and 47 were female. The results of the tests are summarized in Tables 2–18.

TABLE 2. RELATED CONDITIONS IN 100 MONOFIXATORS

| Conditions | Number of patients |
|---|---|
| Corrected strabismus | 66 |
| Anisometropia | 6 |
| Corrected strabismus and anisometropia | 8 |
| Macular lesion | 1 |
| Primary monofixation | 19 |

TABLE 3. TYPE OF STRABISMUS AMONG 74 STRABISMIC PATIENTS

| Corrected strabismus | Corrected strabismus and anisometropia |
|---|---|
| Acquired ET 54 | Acquired ET 7 |
| Acquired XT 6 | Acquired XT 1 |
| Congenital ET 6 | |

Ophthalmoscopic examination revealed pathologic findings in only one patient. The macula of this patient's left eye was completely replaced with a sharply bordered, oval, pigmented lesion, which was slightly larger than one disk in diameter. Two smaller similar lesions were located in the posterior pole of the right eye, but the macula was spared. Ophthalmoscopically, they were typical of the posterior pole chorioretinal lesions in congenital toxoplasmosis. The Toxoplasma

*American Optical Company.
†American Optical Company Catalog No. 11245 Adult Slide.
‡Wirt Stereotest and Stereo Fly Test.

TABLE 4. COMPARISON OF AVERAGE AGE OF ONSET OF STRABISMUS TO AVERAGE AGE OF 74 PATIENTS AT TIME OF STUDY

| Type of strabismus | Number of patients | Average age of onset | Average age at time of study |
|---|---|---|---|
| Acquired ET | 61 | 2 1/2 years | 11 1/2 years |
| Acquired XT | 7 | 1 1/2 years | 12 years |
| Congenital ET | 6 | At birth | 11 1/2 years |

TABLE 5. COMPARISON OF AVERAGE AGE OF DISCOVERY OF ANISOMETROPIA, MACULAR LESION, OR PRIMARY MONOFIXATION TO AVERAGE AGE AT TIME OF STUDY

| Conditions | Number of patients | Average age of discovery | Average age at time of study |
|---|---|---|---|
| Anisometropia | 6 | 7 years | 9 1/2 years |
| Macular lesion | 1 | 5 years | 12 years |
| Primary monofixation | 19 | 8 years | 12 years |

Sabin methylene-blue titers in both patient and mother were 1:4096 when the patient was eight years of age. No other congenital toxoplasmosis stigma was evident.

## DISCUSSION

Since the patients constituting the monofixation syndrome are associated with strabismus, anisometropia, a unilateral macular lesion, or an inherent inability to fuse similar images on each macula, it is helpful to consider each of these conditions as a separate etiologic factor. A patient may arrive at this syndrome by any of these four avenues, or any combination of them.

### ETIOLOGY

This study discloses that the largest single group with the monofixation syndrome consists of strabismic patients who have attained final treatment status. Of the 74 patients in this category, however, eight had anisometropia in addition to strabismus, according to the arbitrary values used in this study to define anisometropia. It is debatable whether a slighter refractive difference between the two eyes than the values selected for defining anisometropia is a significant factor when combined with strabismus in causing the monofixation syndrome. Regardless of how this issue is resolved, strabismus alone ranks as a

TABLE 6. TYPE OF THERAPY FOR 90 PATIENTS WHO RECEIVED TREATMENT

| Condition | Number of patients | Occlusion | Glasses | Miotics | Surgical intervention | Orthoptics |
|---|---|---|---|---|---|---|
| Strabismus, with or without anisometropia | 74 | 45 | 62 | 4 | 43 | 5 |
| Anisometropia | 6 | 4 | 6 | | | |
| Primary monofixation | 10 | 5 | 6 | | | 1 |

TABLE 7. SUMMARY OF 8 ECCENTRIC FIXATION PATIENTS

| Patient number | Strabismus, anisometropia, macular lesion, or primary | Refraction | Ages of occlusion therapy | Best recorded vision in amblyopic eye | Elapse in years since last occlusion therapy | Current vision | Visuscopy |
|---|---|---|---|---|---|---|---|
| 7 | Acq ET | +0.75/+0.50×180 | 2 thru 8 | | 5 | | |
| | | +1.00 | | 20/30 | | 20/50 | Perifoveal |
| 10 | Unilat mac les | −0.50 | None | | | | |
| | | −0.50 | | 2/200 | | 2/200 | Paramacular |
| 13 | Acq ET & aniso | +6.75 | 7 thru 8 | | 2 | | |
| | | +6.25/+2.00×90 | | 20/40 | | 20/50 | Perifoveal |
| 30 | Acq ET | +3.25 | 4 thru 8 | | 3 | | |
| | | +4.00/+0.50×180 | | 20/30 | | 20/70 | Perifoveal |
| 42 | Acq ET & aniso | +4.50 | 2 1/2 thru 5 | | 8 | | |
| | | +6.50 | | 20/30 | | 20/80 | Perimacular |
| 69 | Acq ET | +5.25+1.75×80 | 2 thru 7 | | 4 | | |
| | | +5.75+2.50×100 | | 20/20 | | 20/25 | Perifoveal |
| 83 | Primary | +0.37 | | 20/20 | | 20/40 | Perifoveal |
| | | +0.37 | 6 thru 8 | | 6 | | |
| 93 | Acq ET | +3.50/+2.25×95 | | 20/30 | | 20/50 | Perifoveal |
| | | +3.75/+1.75×75 | 3 1/2 thru 8 | | 5 | | |

TABLE 8. VISUAL ACUITY DIFFERENTIAL BETWEEN RIGHT AND LEFT EYES BY SNELLEN METHOD AT TIME OF STUDY

| Visual acuity | Corrected strabismic patients | Anisometropic patients | Corrected strabismic & anisometropic patients | Macular lesion patient | Primary monofixation patients |
|---|---|---|---|---|---|
| Equal Acuity | 28 | 2 | 2 | | 5 |
| 1 Line Diff | 13 | 1 | 1 | | 3 |
| 2 Line Diff | 11 | 1 | 3 | | 4 |
| 3 Line Diff | 8 | 2 | | | 5 |
| 4 Line Diff | 5 | | 1 | | 1 |
| 5 Line Diff | | | | | 1 |
| 7 Line Diff | 1 | | 1 | | |
| Greater Diff | | | | 1 | |

TABLE 9. DISTRIBUTION OF PATIENTS WITH EQUAL VISION IN BOTH EYES WITHOUT OCCLUSION THERAPY VERSUS THOSE WITH POOR VISION IN ONE EYE

| Number of Patients | Condition | Patients with equal vision in both eyes | Patients with poor vision in one eye |
|---|---|---|---|
| 66 | Corrected strabismus | 17 | 49 |
| | Acquired ET (54) | (11) | (43) |
| | Acquired XT ( 6) | ( 2) | ( 4) |
| | Congenital ET ( 6) | ( 4) | ( 2) |
| 6 | Anisometropia | | 6 |
| 8 | Corrected strabismus and anisometropia | 1 | 7 |
| | Acquired ET ( 7) | | ( 7) |
| | Acquired XT ( 1) | ( 1) | |
| 1 | Macular lesion | | 1 |
| 19 | Primary monofixation | 4 | 15 |

major factor in causing this entity since this study reveals that the two eyes in many strabismic patients have identical, or nearly identical, refractions.

The monofixation syndrome occurs with significantly greater frequency in corrected esotropia than in corrected exotropia.[10,11,24-27] Of the 74 strabismic patients with this entity after the angle of deviation was reduced to straight, or nearly straight, 67 (90 per cent) were esotropes, whereas only 7 (10 per cent) were exotropes. This percentage is not in accord with the usual distribution of successfully treated esotropia and exotropia.

Congenital esotropes appear to have a different reason for monofixating despite the fact that peripheral fusion was acquired by early surgical elimination of the deviation. As has already been suggested by Ing and associates[18] and re-emphasized by Bair[4] and Parks,[43] congenital esotropes seem to have an inherent inability to bifixate. Some congenital esotropic patients obtain peripheral fusion if the eyes have been straightened by surgical intervention at an early age, but they never obtain bifixation. It is tempting to speculate why bifixation does not develop in these patients even though the esotropia is surgically corrected by six months of age. Perhaps there is some justification for Worth's[53] suggestion that these children have a deficit in the fusion faculty. Proof that peripheral fusion is attained by early surgical intervention in a high percentage of congenital esotropes partially discredits this concept (Ing et al.,[18] Taylor[49]). There may be some merit in Worth's thesis, however, since a defect in the central fusion faculty remains a distinct possibility. Another explanation might

be that the eyes must be straightened prior to six months of age if an opportunity is to be provided for bifixation to develop. Regardless of how this observation is explained, it is apparent that none of the therapeutic regimens offered the infant with congenital esotropia to date has produced bifixation. Accordingly, there were 6 monofixators within the group of 100 patients in this recent study who were congenital esotropes and whose eyes were straightened prior to 18 months of age.

Anisometropia is another etiologic factor since it presents an additional obstacle to macular fusion. A clear image on one macula and a blurred image on the other offers little reward for the effort involved to integrate the two into a unified perception. Presuming that similarly clear macular images are required during infancy for establishment of bifixation, one realizes that discovery of anisometropia at an older age is too late to expect bifixation to result from prescription of optical correction for equally clear images on each macula. Unless strabismus also is present, it is difficult to discover the anisometropia during infancy. This study reflects that fact since the six anisometropic patients without strabismus were brought under treatment at a later age than the eight anisometropic patients with strabismus. The question naturally follows, at what age must anisometropia be optically treated to permit bifixation to develop? Unfortunately, too few facts are available for me to answer this question.

The destructive unilateral macular lesion is the organically defective visual counterpart to the unilateral functional macular scotoma that occurs in the binocular visual field of the patient with the monofixation syndrome. It is noteworthy that many patients with only one functioning macula retain straight eyes; this apparently is accomplished by peripheral fusion. For all practical purposes, the binocular vision of patients with an organic cause for the scotoma is indistinguishable from that of those with a functional scotoma.

Patients with primary monofixation are a challenging group to study. It was amazing to discover such a large group of symptomless patients, unaware of the absence of bifixation, unsuspected before disclosure by examination. I became aware of this group originally while studying the stereoacuity among supposedly normal patients. In a successive study (Parks and Eustis[45]) of 793 children over five years of age, without strabismus, anisometropia, or a macular lesion, who appeared for routine ophthalmologic examination in my office, the stereoacuity response indicated that several of them monofixated. A monocular 3° facultative scotoma by binocular perimetry in the visual

field of one or the other eye was confirmed in all. There may or may not have been a small shift to take up fixation by the non-fixating eye upon covering the fixating eye. These patients were totally unaware of any disorder, and without a sophisticated testing technique the examiner, too, would be unaware of its existence. This disorder is not uncommon, which is surprising since heretofore, except for Lang,[27,28] it has been relatively unappreciated as an ophthalmologic entity.

Patients with primary monofixation have a small degree of amblyopia in the non-preferred eye unless fixation is alternated (Parks and Eustis[45]). Four of the nineteen patients in this study were free of amblyopia.

Both congenital esotropic patients and primary monofixating patients seem to have similar inherent defects that prevent central fusion. The latter seem unable to develop bifixation even though their eyes are straight and they have peripheral fusion, which is true likewise for the 6-month-old infant whose congenital esotropia has been surgically corrected. Due to the experience of frequently observing this syndrome in parent, child, and siblings of both congenital esotropic patients and primary monofixators, I wonder if the central fusion defect is not predetermined in these patients, as suggested by Lang.[27,28] The monofixation pattern was recently demonstrated in 12-year-old monozygotic twin boys with almost identical quantities of myopic astigmatism; each had 2 prism diopters of esodeviation by simultaneous prism and cover test with minimal amblyopia in the left eye. More recently, monozygotic 9-year-old triplets were tested. All had (*a*) identical hypermetropia, (*b*) a slightly high accommodative convergence-accommodation ratio (AC/A), (*c*) no deviation by cover-uncover, but a small esophoria elicited by alternate cover at distance which was significantly larger for near, and (*d*) no amblyopia. However, monofixation was proved in all three by binocular scotometry, Bagolini striated lenses, Worth four lights, polarized letters, and poor stereoacuity. Three of four siblings in this family were sufficiently mature to test and all were bifixators. The mother was also a bifixator; the father was not tested.

### CLINICAL CHARACTERISTICS

Several authors have described the patient with the monofixation syndrome as characteristically being slightly amblyopic.[2,12,13,20,22,27,28,32,44,45] The results of this study confirm that the majority of these patients do have amblyopia, but they show further that the percentage of patients with amblyopia varies according to the etiology. Thirty-four

per cent of congenital esotropes, 67 per cent of acquired esotropes, 73 per cent of primary monofixators, 88 per cent of patients with combined strabismus and anisometropia, and 100 per cent of anisometropes have amblyopia. Therefore, it is incorrect to state that the syndrome of monofixation and peripheral fusion characteristically is associated with amblyopia. It is more accurate to state that a minority of congenital esotropes, a majority of the primary monofixators and acquired strabismic patients, and almost all anisometropes with this syndrome have amblyopia.

Some monofixating patients view the world about them with a trivial deviation of their monofixating eye, ranging from 1 to 8 prism diopters of horizontal and 2 to 3 prism diopters of vertical. Others manifest no detectable deviation of either eye by the cover-uncover test, indicating that during ordinary seeing the eyes are straight. Hence, the object of regard is simultaneously imaged on each fovea and, despite this, one image is ignored because the macula upon which it projects does not function during binocular vision. Since deviation of the non-fixating eye is not invariably present, this sign cannot be used to diagnose all cases of monofixation. If a deviation is proved by one eye moving to assume fixation when the fixating eye is covered, this is sufficient to establish the diagnosis of monofixation. On the other hand, if neither eye moves during the cover-uncover test, either monofixation or bifixation may prevail.

Table 10 reveals a slight shift in one eye by cover-uncover test for 63 per cent of the patients in this study, but 37 per cent moved neither eye. Therefore, it is assumed that only two-thirds of the patients with the monofixation syndrome have a deviation that would allow the diagnosis to be made by the cover test. The percentage of deviation versus no deviation varies significantly according to the etiology of the monofixation. Deviation occurred most frequently in those patients treated for strabismus and least frequently in those with anisometropia. The primary monofixators tested midway between these two groups.

Amblyopia is a significant factor in these patients, which might suggest that all monofixators have a small deviation, but in some perhaps a slight degree of eccentric fixation in the non-preferred eye prevents a fixation movement when the preferred eye is covered. However, Table 11 reveals that only three of the 37 patients who manifest no shift to cover-uncover have eccentric fixation according to visuscopy. Furthermore, visual acuity is equal in 16 of the 37 patients. Of these 16 patients, 14 have never received any occlusion therapy. Therefore, it becomes impossible to accept amblyopia as the cause for

TABLE 10. COMPARISON OF MONOFIXATING PATIENTS MANIFESTING A SHIFT TO COVER-UNCOVER TO THOSE MANIFESTING NO SHIFT*

| Condition | Number of patients | Shift | No shift |
|---|---|---|---|
| Corrected strabismus | 66 | 47 | 19 |
| Anisometropia | 6 | 1 | 5 |
| Corrected strabismus and anisometropia | 8 | 4 | 4 |
| Macular lesion | 1 | | 1 |
| Primary monofixation | 19 | 11 | 8 |

*This test was performed at 20 feet and at 13 inches from the patient. A shift at either distance, or both, was scored as positive.

TABLE 11. RELATIONSHIP TO TYPE OF FIXATION IN 37 PATIENTS MANIFESTING NO SHIFT TO COVER-UNCOVER

| Condition | Number of patients | Central fixation | Eccentric fixation |
|---|---|---|---|
| Corrected strabismus | 19 | 18 | 1 |
| Anisometropia | 5 | 5 | |
| Corrected strabismus and anisometropia | 4 | 4 | |
| Macular lesion | 1 | | 1 |
| Primary monofixation | 8 | 7 | 1 |

TABLE 12. RELATIONSHIP OF 34 PATIENTS MANIFESTING CENTRAL FIXATION IN EACH EYE AND NO SHIFT TO EITHER COVER-UNCOVER OR ALTERNATE COVER

| Condition | Number of patients | No alternate cover shift | Alternate cover shift |
|---|---|---|---|
| Corrected strabismus | 18 | 13 | 5 |
| Anisometropia | 5 | 4 | 1 |
| Corrected strabismus and anisometropia | 4 | 3 | 1 |
| Primary monofixation | 7 | 5 | 2 |

no shift of the non-fixating eye when the fixating eye is covered in more than a small minority of the monofixation syndrome patients, as Helveston and von Noorden[17] described in their microtropic patients. That approximately one-third of all monofixating patients have straight eyes during binocular vision appears to be a valid conclusion. I deduce from these facts that bifixation can occur only in those patients free of a deviation, but that the absence of a deviation does not guarantee that the patient is bifixating.

Another clinical characteristic often found in patients manifesting a shift to cover-uncover is a greater deviation by prism and alternate cover than by simultaneous prism and cover. Occasionally the differ-

TABLE 13. RELATIONSHIP OF SIMULTANEOUS PRISM AND COVER MEASUREMENTS TO ALTERNATE COVER MEASUREMENTS IN 63 PATIENTS MANIFESTING A SHIFT TO COVER-UNCOVER

| Condition | Number of patients | Alternate cover unchanged | Alternate cover increased |
|---|---|---|---|
| Corrected strabismus | 47 | 27 | 20 |
| Anisometropia | 1 | 1 | |
| Corrected strabismus and anisometropia | 4 | 4 | |
| Primary monofixation | 11 | 5 | 6 |

ence in quantity is striking – 10 prism diopters or more. Table 13 reviews this feature and shows that 26 of the 63 patients with a shift to cover-uncover had an increase in the angle of misalignment when fusion was prevented during the alternate cover test. The probable reason for this phenomenon is the benefit derived by the patient who reduces the angle of deviation with his fusional vergence.

Binocular single vision is divided into central and peripheral fusion, depending on whether or not the cerebrally integrated similar retinal images in each eye project onto foveal or extrafoveal areas. Differences between central and peripheral fusion are more than can be accounted for simply in resolving power between fovea and extrafoveal retina. For example, both Linksz[30] and Adler[1] discussed the fact that dissimilar retinal images projecting onto the foveas are not simultaneously perceived, but those projecting onto the extrafoveal areas are. This implies a very fundamental difference in the physiology of fusion between rod-free and rod-populated retinal areas. As Adler stated, there is no question about simultaneous perception of images projecting onto the rod-populated retinal areas, and everyone except Verhoeff[50] seems to take it for granted that there is true fusion of similar images on the foveas. Verhoeff claims that, instead of foveal images being fused, there is rapid alternation of portions of one image with the other in retinal rivalry. Another difference between central and peripheral fusion is the fact that peripheral fusion is obtainable with a greater deviation than that permitted by central fusion since Panum's fusional space expands as it proceeds peripherally away from the fixation point.

Clinically at least, it appears that central fusion is always accompanied by peripheral fusion, but the reverse is not true. Central fusion seems to offer nothing additional to fusional vergence amplitudes over and above that provided by peripheral fusion alone, since the fusional vergence amplitudes of monofixators equal those of bifixators.

A characteristic of the young heterotropic patient who has the capacity for binocular single vision is adaptation of the sensorial status to conform to the abnormal motor status. Central fusion is invariably forfeited, if ever it was present, prior to the onset of the heterotropia, since the foveas receive dissimilar images that cannot be simultaneously perceived. The similar images on the fixating eye's fovea and the non-fixating eye's extrafoveal area are simultaneously perceived, causing the object of regard to be diplopic unless one image is suppressed. Apparently, suppression is the ultimate adaptation for the central portion of the binocular visual field in strabismus. It is a cortical inhibitory reflex that (*a*) gradually develops in depth of proficiency with the duration of the strabismus, (*b*) is localized to the deviated eye's retinal area, which receives the same image material projected upon the fixating eye's macula, and (*c*) is constantly changing its retinal location as the angle of strabismus is altered. The suppressed retinal area projects a scotoma out into the binocular visual field that surrounds the object of fixation.

The sensorial adaptation to the motor abnormality of strabismus is entirely different in the peripheral portion of the binocular visual field than the adaptation for the central portion. Instead of eliminating the peripheral visual field of one eye as occurs in suppression, the peripheral binocular visual field perpetuates itself by cortically rearranging the innate directional values of the retinal neuroepithelial elements to eliminate the diplopia. The normal corresponding directional values of the anatomically corresponding neuroepithelial elements of the two retinas are altered, and this readjustment is referred to as ARC.* Hence, the ultimate adaptation to the diplopia in the peripheral binocular

*The classic thesis of Burian[8,10,11] on ARC, and description of ARC by Lancaster[26] encourage the incomplete concept about ARC. Burian and other authors approach the subject by first describing NRC, illustrating it by referring to the foveas as corresponding retinal points, possessing identical directional values, upon which the fixation point is imaged, and naturally fused since diplopia is impossible. The strabismic patient, however, according to the classic explanation of ARC, experiences diplopia of the fixation point as it projects onto the fovea of the fixating eye and extrafoveal area of the deviating eye unless a shift converts their directional values from dissimilar to identical. With this explanation the student concludes that ARC is a sensory adaptation to the strabismic angle, confined to the retinal areas of two eyes upon which projects the fixation point. Actually, in small print, Burian[10] cites Kretzschmar's[24,25] evidence that ARC exists over the whole of the binocular field – at least when ARC is well established. Burian does allude to suppression occurring in ARC, but in a manner that is insufficient to destroy the concept that the fixation point is fused in ARC. The Bagolini striated lens method of investigating ARC, however, leaves no doubt that the fixation point in a strabismic patient with ARC is suppressed in the non-fixating eye and, actually, its surrounding retina along with the extramacular retina in the fixating eye manifest the ARC.

field caused by the strabismus is to change the NRC to ARC and continue binocular single peripheral vision.

Suppression and ARC develop concomitantly in a strabismic child. Development of suppression is apparently the solution for the annoyance of diplopia in the central portion of the binocular visual field, but perpetuation of binocular single peripheral fusion with ARC is the solution for the diplopia in the peripheral binocular field. Therefore, even in adapting the sensory physiology to the motor abnormality, there is a difference between central and peripheral fusion.

The foregoing explanation of suppression and ARC emphasizes that sensorial adaptations develop in the young child in response to the annoyance of diplopia caused by the strabismus. A motor response to the same stimulus would be application of sufficient fusional vergence to offset the deviation, but generally this is a practical solution to the diplopia problem only in the patient with a small deviation. The fusional vergences, however, are easily capable of overcoming the trivial deviation found in the monofixation syndrome. Furthermore, some patients with this syndrome have no deviation even to alternate cover. Yet, whether there is no deviation or a trivial deviation up to 8 prism diopters during binocular seeing, a retinal suppression area that includes the macula is present in the non-fixating eye. This causes a scotoma to be projected into the central region of the binocular visual field, destroying any opportunity for central fusion. At least in the patient whose non-fixating eye is not deviated, the suppression area cannot be attributed to a sensorial adjustment to diplopia. Even in patients who have a trivial deviation it is difficult to accept the idea that the suppression is an adaptation to diplopia, since the fusional vergence amplitudes exceed the small deviation by a comfortable margin. Actually in those patients having a deviation, it appears that the suppression contributes to, rather than results from, the deviation. Supporting this concept is the fact that 26 of the 63 patients in this study who had a deviation to cover-uncover partially reduced their alternate cover deviation (Table 13), and they could probably have reduced it completely were it not for the location of the retinal suppression. If these patients were obtaining relief from diplopia by use of suppression, why wouldn't they develop the suppression in the region of the retina that conformed to their angle of deviation elicited by alternate cover?

Lack of knowledge of the pathophysiology involved in the monofixating syndrome allows only conjecture to explain the invariable functional central retinal suppression in all patients except those with

a destructive macular lesion. The concept that seems most reasonable emphasizes the following:

1. Peripheral and central fusion are separate physiologic entities.
2. Peripheral fusion is less complex and more easily acquired in infancy.
3. Some patients seem to have a genetically determined inability to acquire central fusion despite having peripheral fusion, straight eyes, and no anisometropia.
4. Congenital strabismus and congenital anisometropia are factors that interfere with development of central fusion.
5. Both acquired strabismus and acquired anisometropia interfere with the continuance of central fusion, and the prognosis of its restoration is inversely related to the duration of these interfering factors.

According to the strict definitions of NRC and ARC, peripheral fusion is achieved in straight eyes with NRC and in deviated eyes with ARC. Therefore, the retinal correspondence in the monofixation syndrome patients varies according to the presence or absence of a deviation by cover-uncover. Actually in these patients with such trivial deviation, I question whether the neurophysiologic processes required to shift retinal correspondence from NRC to ARC really occurred. Could it be that the definition of ARC is too strict? Jampolsky[20] suggested that there may be a stretched-out Panum's area to allow these patients continuance of fusion. This would make the neuropathophysiologic adaptation of ARC unnecessary. The physiology involved is probably looser than the tight definition of NRC fusion, which excludes it in a patient with deviation and fusion. Possibly the NRC physiology is sufficiently elastic to allow peripheral fusion in patients having deviation up to 8 prism diopters. Regardless of whether it is NRC or ARC that permits peripheral fusion in patients with deviation in this syndrome, their sensory responses to testing for binocular single vision are indistinguishable from those patients without deviation.

Unquestionably the patients under discussion possess simultaneous perception, fusion with fusional vergence amplitudes, and stereopsis appreciation. These three facets of binocular single vision can be tested by several methods. Certain inherent difficulties in this particular group of patients limit use of the major amblyoscope to investigate all three facets. The sensory status of these patients is best investigated by using techniques that simulate everyday seeing. The major amblyoscope does not satisfy this stipulation because the patient must look down tubes; it is too cumbersome to provide minute de-

tailed investigations of very small angle deviations; and the slide selection available for use with this instrument does not permit the most accurate determination of the fusional vergence amplitudes and the stereoacuity. For these reasons, the major amblyoscope was not used for this study.

Worth's four lights provide immediate information about peripheral fusion and presence of a scotoma in the binocular visual field of the monofixating patient. It is practicable for use with children as young as four years of age. Instruction is not required before the test is performed. A response that four lights are seen is accepted as evidence of fusion, and the statement by many that the white light is seen in red-green rivalry is additional confirmation of fusion. However, the examiner must be aware that possibly the lights projecting onto the retina of the non-fixating eye are entirely within the suppression area. Since most of the scotomas in the binocular field of the monofixating patients are approximately 3 degrees, the Worth lights are not fused at distance but they are at near. The distance Worth four lights project more peripherally onto the retinas as the patient approaches; the negative response for fusion suddenly changes to positive at the point at which the lights first project outside the scotoma. None of the 100 patients in this study fused the distant Worth lights, but all fused them between 2 and 18 feet; the average patient approached to within 10 feet before fusion occurred.

The near Worth four lights held at 13 inches were fused by 99 of the 100 patients; only the patient with the macular lesion did not obtain fusion. This is verification that the non-functioning central retinal area in his involved eye is larger than the retinal suppression areas in the other monofixators. Not until the near lights were brought to within 6 inches did he fuse; this patient also had to approach to within 2 feet of the distant Worth lights before fusion occurred. His retinal lesion was slightly larger than 1 disk diameter in size. It can be proved that a scotoma exists in the binocular field of all monofixators by moving the near Worth four lights beyond 13 inches until fusion disappears. Most of the monofixating patients lose their fusion for the near Worth lights as the lights retrocede to a point between 3 and 5 feet from the patient. When the lights are moved back toward the patient, he suddenly appreciates fusion once again as the lights move outside the scotoma.

This use of either the distant or near Worth four lights is particularly suitable for testing fusion and suggesting the presence of a scotoma in the non-fixating eye of the monofixator. It can also demon-

strate whether the patient is capable of switching fixation and transferring the scotoma to the visual field of the opposite eye. If the size of the angle of projection of the lights is smaller than the size of the scotoma, the patient will see either two red or three green lights, depending on whether the non-fixating eye is behind the red or the green filter. He is requested to look in the direction opposite to whichever lights he has seen, and then it is determined whether the first lights disappear. Patients who have a strong preference for fixation with one eye find it extremely difficult, or impossible, to hold fixation with the non-preferred eye. It is impossible in such patients to demonstrate that the scotoma can be transferred to the visual field of the opposite eye.

The quality of fusion reported from patients with a deviation measuring up to 8 prism diopters by cover-uncover is no different than in those patients with no deviation. The definitions of NRC and ARC make it obligatory to record the fusion response in the deviator as ARC and the fusion response in the non-deviator as NRC, but does the fusion response to the Worth four lights represent something fundamentally different in the sensorium of the patient having deviation of the non-fixating eye up to 8 prism diopters compared to the one who has no deviation?

The Bagolini striated lens test is the best method for evaluating the retinal correspondence in the monofixation syndrome. It offers an advantage to the examiner who can observe the finding of the cover-uncover test while the patient fixates the light source of the streaks he perceives in his binocular visual field. This permits the examiner to relate the alignment of the patient's eyes, objectively determined by the cover-uncover test, with the subjective response of the location of the streaks in reference to the light. Regardless of the presence or absence of a deviation, the answer was identical from all the monofixation syndrome patients in response to the question about location of the streaks in reference to the light. Each claimed only one light was seen and the streak seen by each eye passed through the light. The interpretation of this response, related to the objectively determined alignment, is that all non-deviators have NRC and all deviators have ARC according to the definitions of NRC and ARC. However, I wonder whether the application of our strict definitions of NRC and ARC to this group of patients is not more harmful than helpful. It conditions us to reason that, in the patients having a deviation, a pathologic process has developed in the sensorium that serves retinal correspondence so binocular single vision may continue.

The range of the horizontal fusional vergence amplitude for the monofixators in this study is similar to that found in any group of bifixators. Among the monofixating patients, there is no difference in the average fusional vergence amplitudes between those who fuse without deviation (NRC) and those who fuse with a deviation of 8 prism diopters or less (ARC). This is in sharp contrast to the fusional vergence amplitude found in strabismic patients having greater than 8 prism diopters of deviation and ARC, which is usually very limited or non-existent.* These facts lend further support to the possibility that the current definition of ARC leads us to incorrect reasoning about the retinal correspondence in the monofixating, peripherally fusing, patients with a deviation ranging up to 8 prism diopters. In these patients is there an actual change in the neurophysiologic process that serves NRC to something different that serves ARC? It could be that the peripheral fusion with NRC is sufficiently loose to allow a deviation up to 8 prism diopters with no change in neurophysiology.

The monofixation syndrome patients do appreciate stereopsis, but obtain this perception only from relatively large degrees of horizontal retinal image disparity compared with the excellent stereoacuity of bifixators. Burian[8] states that stereopsis does not come about through horizontal disparity on the basis of an anomalous retinal relationship. My clinical experience in obtaining stereopsis in ARC patients with heterotropia larger than 8 prism diopters, or in those who manifest no simultaneous perception capability regardless of their alignment, corroborates Burian's statement. At least a horizontal retinal image disparity of 3000 seconds of arc is insufficient in these patients to evoke a stereopsis response. Yet, in the so-called ARC patients with 8 prism diopters or less of deviation who can fuse Worth four lights, demonstrate a fusional vergence amplitude, and simultaneously perceive the streaks on each retina created by Bagolini striated lenses, stereopsis is invariably demonstrated. Can this be further evidence that according to the current definition of ARC, ARC is semantically correct for the monofixation syndrome patients with a small tropia by cover-uncover but physiologically NRC peripheral fusion is actually present?

Polaroid vectographs offer a convenient, accurate, and simple

*Burian[7] elicited fusional movements in strabismic patients with ARC using the peripheral areas of the retinas to obtain the response, but Hallden[16] suggested that the angle of anomaly may shift in response to the image movement across the retinas, simulating fusional movements, in the patients with ARC. This suggestion is refuted simply by the observation that the eyes actually move during vergence testing with the rotary prism and suddenly return toward the zero position when fusion is broken.

TABLE 14. TABULATION OF STEREOACUITY OF 100 PATIENTS ACCORDING TO THE WIRT STEREOTEST

| Condition | Number of patients | Stereoacuity at 16 inches in seconds of arc | | | | | |
|---|---|---|---|---|---|---|---|
| | | 3000 | 1000 | 400 | 200 | 100 | 67 |
| Corrected strabismus | 66 | 3 | 11 | 13 | 23 | 12 | 4 |
| Anisometropia | 6 | | | | 2 | 4 | |
| Corrected strabismus and anisometropia | 8 | | 1 | | 3 | 1 | 3 |
| Macular lesion | 1 | | | | | 1 | |
| Primary monofixation | 19 | | 1 | 5 | 6 | 4 | 3 |

method for determining stereoacuity. The stereoacuity of the monofixators in this study ranged from 67 to 3000 seconds of arc as shown in Table 17. A previous study of monofixators in treated strabismic patients by Parks and Eustis[45] revealed the identical range of stereoacuities. By contrast, one of the attributes of bifixation is superb stereoacuity. A study of 596 normal subjects with proved bifixation revealed their stereoacuity to range between 14 and 40 seconds of arc, with an average of 24 seconds of arc.[45]

In this study, the average stereoacuity is the same in the 100 monofixators who are orthophoric in comparison with those who have a deviation by cover-uncover. Apparently the basic issue in determining the stereoacuity is the scotoma in the visual field of the non-fixating eye. Bifixation allows the high resolving powers of each macula to detect minute degrees of retinal image disparity; hence, the stereoacuity is good. In monofixation the retinal image disparity is detected by studying the images on retinal areas having low resolving power, which causes poor stereoacuity. Consequently, the same retinal areas with poor resolving power are used to determine stereoacuity in either monofixator, with or without deviation. Derived from these facts, stereoacuity previously has been reported as a very reliable indicator of either monofixation or bifixation.[41]

A polaroid vectographic test is more limited in the range of retinal image disparities offered the patient than the near stereoacuity tests. The correlation between the distance and near stereoacuities in this study was poor. The best stereoacuity was 120 seconds of arc for distance and 67 seconds of arc for near. In fact, only 29 patients had 240 seconds of arc or better at distance as shown in Table 18. By comparison, Table 17 shows that 66 patients had 200 seconds of arc of stereoacuity or better at near.

Both the near stereoacuity and distant stereoacuity vectographic techniques are equally simple for use by the examiner. The time

TABLE 15. TABULATION OF STEREOACUITY OF 100 PATIENTS ACCORDING TO THE POLAROID VECTOGRAPHIC SLIDE

| Condition | Number of patients | Stereoacuity at 20 feet in seconds of arc | | | |
|---|---|---|---|---|---|
| | | Less than 240″ | 240″ | 180″ | 120″ |
| Corrected strabismus | 66 | 52 | 4 | 2 | 8 |
| Anisometropia | 6 | 1 | 1 | | 4 |
| Corrected strabismus and anisometropia | 8 | 4 | 2 | | 2 |
| Macular lesion | 1 | 1 | | | |
| Primary monofixation | 19 | 13 | 1 | 2 | 3 |

demanded of the examiner to give either test is identical. In the routine evaluation of the patient, it is redundant to measure the stereoacuity at both distance and near. The most reliable of the various stereoacuity tests available at present is the Wirt near stereoacuity test; it provides the most distinct separation between monofixators and bifixators.

Much attention has been directed to the absolute scotoma in the non-fixating eye, since it is the single invariable sensory finding in the monofixation syndrome. The scotoma facultatively disappears within the non-fixating eye when its monocular visual field is plotted unless an organic retinal disorder is the cause of the monofixation syndrome. The scotoma is absolute inasmuch as nothing is seen within this area as long as fixation is maintained in the opposite eye. Most scotomas vary from 3 to 5 degrees in horizontal dimension and slightly less in the vertical meridian. Occasionally, the scotoma will extend a degree or two further onto the nasal retina in the monofixating esodeviators and slightly more onto the temporal retina in the monofixating exodeviators.

The red-green filters used for binocular scotometry dissociate the eyes in the darkened room. Hence, the location of the scotoma in reference to the fixation target is positioned according to the deviation of the eyes disclosed by alternate cover. In orthophoric patients, the scotoma is centered around the fixation target; in esodeviators, it is displaced heteronomously; and in exodeviators, it is displaced homonomously. In patients with the monofixation syndrome NRC is invariably demonstrated with this test.

Bifixators have a response to binocular perimetry dramatically different from that of monofixators. The bifixator superimposes the green test target upon the fixation target without hesitation. Unless the patient is orthophoric, however, the test target actually is displaced

TABLE 16. RESULTS OF ATTEMPT TO PLOT THE SCOTOMA IN BOTH THE RIGHT AND LEFT VISUAL FIELD OF 100 PATIENTS BY BINOCULAR PERIMETRY

| Condition | Number of patients | Scotoma plotted in either eye | Scotoma plotted in one eye | Scotoma plotted in neither eye |
|---|---|---|---|---|
| Corrected strabismus | 66 | 49 | 16 | 1 |
| Acquired ET | (54) | (40) | (14) | |
| Acquired XT | ( 6) | ( 4) | ( 2) | |
| Congenital ET | ( 6) | ( 5) | | (1) |
| Anisometropia | 6 | | 6 | |
| Corrected strabismus and anisometropia | 8 | 5 | 3 | |
| Acquired ET | ( 7) | ( 4) | ( 3) | |
| Acquired XT | ( 1) | ( 1) | | |
| Macular lesion | 1 | | 1 | |
| Primary monofixation | 19 | 10 | 9 | |

TABLE 17. RESULTS OF ATTEMPT TO ELICIT THE SCOTOMA IN BOTH THE RIGHT AND LEFT EYES OF 100 PATIENTS BY BAGOLINI STRIATED LENS TEST

| Condition | Number of patients | Scotoma elicited in either eye | Scotoma elicited in one eye | Scotoma elicited in neither eye |
|---|---|---|---|---|
| Corrected strabismus | 66 | 43 | 17 | 6 |
| Acquired ET | (54) | (34) | (14) | (6) |
| Acquired XT | ( 6) | ( 4) | ( 2) | |
| Congenital ET | ( 6) | ( 5) | ( 1) | |
| Anisometropia | 6 | | 6 | |
| Corrected strabismus and anisometropia | 8 | 3 | 5 | |
| Acquired ET | ( 7) | ( 2) | ( 5) | |
| Acquired XT | ( 1) | ( 1) | | |
| Macular lesion | 1 | | 1 | |
| Primary monofixation | 19 | 8 | 10 | 1 |

from the fixation target according to the point at which the visual axis of the non-fixating eye strikes the screen when superimposition of the targets is claimed. In contrast, the monofixators manifest frustration as the test target disappears during its approach toward the fixation target.

The scotoma can be plotted by the red-green projector and filter technique in almost all patients having the monofixation syndrome. The scotoma is probably always in the visual field of the non-fixating eye, but some patients find it impossible to hold fixation of the non-preferred eye on the fixation target as the test target approaches it. As the test target reaches the scotoma boundary, these patients surrender to the compulsion to switch fixation from the fixation target to the test target. Instead of the test target being within the scotoma, the fixation target is now located there, and any opportunity to plot

TABLE 18. RESULTS OF THE 4Δ BASE-OUT TEST OF 100 PATIENTS FOR CONFIRMATION OF A SCOTOMA

| Condition | Number of patients | Positive response to 4Δ base-out test | Questionable response to 4Δ base-out test |
|---|---|---|---|
| Corrected strabismus | 66 | 44 | 22 |
| Acquired ET | (54) | (39) | (15) |
| Acquired XT | ( 6) | ( 4) | ( 2) |
| Congenital ET | ( 6) | ( 1) | ( 5) |
| Anisometropia | 6 | 6 | |
| Corrected strabismus and anisometropia | 8 | 6 | 2 |
| Acquired ET | ( 7) | ( 6) | ( 1) |
| Acquired XT | ( 1) | | ( 1) |
| Primary monofixation | 19 | 15 | 4 |

the scotoma in the non-preferred eye has been lost. This inability to hold fixation with the non-preferred eye was manifest in 35 of the patients in this study and, as a result, the scotoma was demonstrable for these patients only in the non-preferred eye (Table 14). Of the 35 patients who found it impossible to sustain fixation of the fixation target with the non-preferred eye, 32 were amblyopic and 8 of these were eccentric fixators. However, 31 amblyopic patients could maintain fixation of the fixation target with their amblyopic eye, permitting the scotoma to be plotted in their non-amblyopic eye. Amblyopia was minimal in all 31 patients; the poorest vision was 20/40. Amblyopia is, therefore, a definite factor that interferes in maintaining fixation of the fixation target with the non-preferred eye.

The Bagolini striated lens test is another technique for disclosing the invariable scotoma in the visual field of the non-fixating eye in monofixators. The streaks extend out into the periphery far beyond the suppressed retinal area. A little more of the streak on one side or the other of the light may be missing, and this is somewhat related to the deviation. Often, more of the streak projected onto the nasal retina is missing in esodeviators, whereas more of the streak projected onto the temporal retina is missing in exodeviators. The gap around the fixation light, projected onto a grid, measures approximately a 3- to 5-degree scotoma.

It is more difficult to prove the presence of a scotoma by the striated lens test than by binocular perimetry. The test is too difficult for use with children under eight years of age. Table 16 indicates that 7 of the 100 patients in this study were not aware of a scotoma. The scotoma was elicited in either eye of 54 patients as fixation was switched from right to left, but in only one eye of 39 patients, because

TABLE 19. COMPARISON OF 4Δ BASE-OUT TEST RESPONSE IN PATIENTS MANIFESTING A SHIFT TO COVER-UNCOVER TO THOSE MANIFESTING NO SHIFT

| Cover-uncover test | Number of patients | 4Δ base-out test positive response | 4Δ base-out test negative response |
|---|---|---|---|
| Shift | 55 | 52 | 3 |
| No shift | 45 | 20 | 25 |

TABLE 20. THE 4Δ BASE-OUT TEST RESPONSE RELATED TO THE PRESENCE OF AMBLYOPIA IN THE 45 PATIENTS MANIFESTING NO SHIFT TO COVER-UNCOVER

| 4Δ base-out test | Number of patients | Amblyopia | No amblyopia |
|---|---|---|---|
| Positive response | 20 | 20 | |
| Negative response | 25 | 5 | 20 |

some patients were unable to maintain fixation with the non-preferred eye.

Another method frequently used to reveal the scotoma in the monofixation syndrome is the 4-diopter base-out prism test, but a scotoma was proved in only 72 of the 100 patients by this test (Table 19). The response in the patients who manifested a shift to cover-uncover to the 4-diopter base-out prism test was generally positive for a scotoma, but the response for a scotoma was often negative in those patients who exhibited no shift to cover-uncover. Table 20 shows that only three of the 55 patients having a shift to cover-uncover responded negatively for a scotoma, while 25 of the 45 patients having no shift to cover-uncover responded negatively. Possibly, the explanation for this observation is the fact that the monofixator, with no shift to cover-uncover, is more apt to switch fixation from one eye to the other when the 4-diopter base-out prism is placed before the fixating eye rather than to refixate this eye after fixation is broken by the sudden prismatic shift of the visual field. This explanation is particularly attractive for those who are not amblyopic. Validity of this explanation is strengthened by the facts shown in Table 21. Of the 45 patients having no shift to cover-uncover, 20 were not amblyopic, and the negative response for a scotoma according to the 4 prism diopter base-out test was found in all 20 of these patients. In only five of the 25 amblyopic patients was there a negative response for the presence of a scotoma. In those patients with a negative response for a scotoma, neither eye tended to shift when the 4-diopter base-out prism was placed before either, although on some occasions, a shift did occur when the prism was placed before one eye or the other. However,

TABLE 21. COMPARISON OF PERCENTAGES OF THE 100 PATIENTS IN WHOM SCOTOMA WAS CONFIRMED ACCORDING TO THE DIAGNOSTIC METHOD USED

| Diagnostic method used | Percentage of patients in whom scotoma was confirmed |
|---|---|
| Worth four lights | 100 |
| Polaroid vectographic slide | 100 |
| Binocular perimetry | 99 |
| Bagolini striated lens test | 93 |
| 4Δ base-out test | 72 |

the response was not repeatable on retesting and the lack of consistency of response seemed insufficient to validly conclude the presence of a scotoma; consequently, the response was scored as negative.

Another method for the study of the scotoma made use of the A-O Vectographic Project-O-Chart Slide. All of the patients responded to testing with this slide by proving they were monofixators, since only the polarized letters projecting into the fixating eye were seen. Each was unaware of the letters projecting into the other eye, presumably due to the presence of the scotoma within the visual field of that eye.

The polarized letters of the polaroid vectographic slide provide a rapid and dependable differentiation of bifixator and monofixator. Although all 100 monofixators deleted the letters projected into the non-fixating eye, five patients claimed that while reading the line some letters appeared while others disappeared. I presume these patients rapidly alternated fixation from macula to macula as the line of letters was studied. This response could mislead the examiner if the patient did not spontaneously comment about the ever-changing letters appearing and disappearing.

Table 21 compares the reliability of the five methods used in this study to detect the invariable scotoma present in the monofixator.

For many reasons, the validity of the current definition of ARC is questioned. In the first place, the clinical definition of ARC currently appears to be based on the Bagolini striated lens test, the binocular visuscope test (bifoveal correspondence test), and the image transfer test.[46] For this study only the Bagolini striated lens test was used because it simulates the ordinary seeing circumstances encountered in binocular vision. The other two are laboratory tests devoid of this virtue. In fact, the binocular visuscope test relates the dissimilar targets on each fovea, a factor which precludes bifoveal simultaneous perception, and only one eye is uncovered during the after-image transfer

test. However, all these tests suggest that ARC[40] is present in the monofixators who have a small heterotropia identified by cover-uncover. The Bagolini striated lens test response of both lines appearing to pass through the light, despite a small inexactness of intersection of the visual axes at the light, can be explained by Panum's fusional space being sufficiently large peripherally to the scotoma to permit the peripheral simultaneous perception with NRC. The deduction that ARC is diagnosed by this response is invalid since this deduction is based on the quantity of inexactness of the visual axes permitted for bifixation (fixation disparity). However, in the monofixation syndrome, the central scotoma changes this value, permitting a much larger quantity of deviation before ARC must be developed to replace NRC in order to achieve peripheral fusion. Therefore, there is a very good possibility that many of the early authors[20,32,44,45] on this subject, who considered the patients with the monofixation syndrome to have NRC, reasoned correctly, and many of the later contributors,[17,28,40] who contend ARC is present, reasoned incorrectly.

The other factors that suggest NRC rather than ARC in monofixators are discussed elsewhere in this paper, but are listed in order here:

1. There is some compelling force that drives the eyes into a position of 8 prism diopters or less of deviation. The ARC adjusts to the angle of the deviation, but in this case often the everyday angle of deviation in the monofixators adjusts to an angle less than that disclosed by alternate cover. If ARC is actually present, why would it not develop for the angle of deviation revealed by alternate cover?
2. The ability to fuse Worth four lights in monofixators with a deviation (supposedly ARC) is identical to those without a deviation (supposedly NRC).
3. There is no difference in the average fusional vergence amplitudes between monofixators with a deviation (supposedly ARC) and those without a deviation (supposedly NRC).
4. Both those patients with a deviation (supposedly ARC) and those without a deviation (supposedly NRC) have identical stereoacuity findings.
5. The central scotoma in monofixators is identical in those with a deviation (supposedly ARC) and those without a deviation (supposedly NRC).
6. Monofixators with a deviation have NRC according to binocular perimetry (a dissociated test) and ARC according to the Bagolini striated lens test (a non-dissociated test).
7. The prognosis for the eyes to remain unchanged in alignment

over the years is the same for both those with deviation (supposedly ARC) and those without deviation (supposedly NRC).

## TREATMENT

The primary objective of treatment is to induce the patient to surrender the scotoma and become attentive simultaneously to the similar images on each macula – to cease monofixating and begin bifixating. However, thus far all attempts to accomplish this objective have met with failure. The treatment can be divided into motor and sensory.

Improvement of the motor problem is not generally necessary, since the maximal deviation in the normal binocular seeing situation never appears to exceed 8 prism diopters; this is usually within the range of being reduced to zero easily by the patient's fusional vergence. Occasionally, the alternate cover deviation in a patient may be a horizontal deviation of 20 prism diopters or more, which causes intermittent diplopia when there is a lapse of the fusional vergence that was maintaining an 8 prism diopter or less cover-uncover deviation. This rare patient may benefit from surgery designed to eliminate the alternate cover deviation. Prismatic correction of the motor imbalance may be used in lieu of surgery, but indications for this procedure are equally rare. Except for the rare case that demands considerable fusion effort to control the large deviation, no benefit is derived from correction of the usually small alternate cover deviation by either surgery or prisms because monofixation persists. The motor imbalance apparently is not the cause of the syndrome.

Inasmuch as these patients have adequate fusional vergence amplitudes, there is rarely a need to prescribe fusional vergence exercises. However, if they are prescribed, the amplitudes increase with the same ease as in the bifixating patients.

Sensory treatment includes monocular and binocular therapy. Monocular therapy essentially is amblyopia treatment. Unless the monofixating child alternates fixation from one macula to the other, the non-preferred eye becomes amblyopic. Occlusion therapy adequately manages this sensory complication. If amblyopia tends to return when occlusion is terminated, partial occlusion is maintained until nine years of age. Occlusion for one-half the day is adequate to prevent restoration of amblyopia. If glasses are worn, the lens before the preferred eye is occluded for half the day. Preferably, two pairs of glasses are provided; those with an occluder lens* before the preferred eye are worn one-half the day, or after school, while those with the clear lenses are worn during the rest of the day.

*American Optical Company Occluder Lens.

Treatment of amblyopia, a monocular sensory defect, does not affect the scotoma, a binocular sensory defect, in the monofixation syndrome. The logical therapeutic approach for overcoming the suppression is training the patient to appreciate diplopia, but my experience has shown that these patients are refractory to learning diplopia recognition other than physiologic diplopia, or diplopia induced by displacing the image outside the suppression scotoma with prisms.

Five patients in this study received antisuppression orthoptic instruction with the intent to teach bifixation. All were minimally esodeviated by cover-uncover. Four had acquired esotropia; in three the deviation was reduced by glasses to the quantity acceptable for monofixation syndrome, and, in the other, surgery was required in addition to the glasses. The fifth patient had primary monofixation syndrome, and the only other treatment was occlusion for amblyopia. All attempts to convert their monofixation habit to bifixation met with complete failure. I have encountered claims that binocular function may be learned by using an orthoptic technique built around the entoptic phenomenon of Haidinger's brushes. However, no documentation of these claims has been published. My experience with this technique has been insufficient to justify comment, but I do caution that successful claims for converting monofixation to bifixation by this treatment may be only the production of rapid alternation of fixation rather than genuine bifixation. A patient treated by this method should be accepted as a bifixation cure only if the stereoacuity has been converted from 67 seconds of arc or less to 40 seconds of arc or better, and no scotoma is evident to the five tests used in this study for detecting a scotoma.

Anisometropic patients may be converted to alternating use of their monofixating maculas by spectacle or contact lens prescription. Supplying equally clear images simultaneously to each macula generally does not improve the chance for bifixation any more than compensating for the small deviation with prism spectacles, but some very rare exceptions to this generalization have been documented.

## PROGNOSIS

The most impressive prognostic feature of patients with the monofixation syndrome is their static alignment state. Over the years their eyes continue to remain aligned as well as if they were bifixating, regardless of the associated factors of strabismus, anisometropia, or a unilateral macular lesion – or absence of all three. The average difference between the ages of reduction of deviation angle to within 8 prism diopters of straight and the final age of the 74 strabismic patients in

this study is approximately 9 years, ranging from 2 to 16 years. The 19 primary patients averaged 12 years of age, the 6 anisometropic patients averaged 9½ years of age, and one patient with a macular lesion was 12 years of age at the time of this study (Table 5); all 26 are presumed to have been monofixators with straight eyes since early infancy. These data reveal the tendency for the alignment of the monofixator to persist unchanged over the years, a fact noted by many other contributors[20,22,28,32,44,45] to this subject. Peripheral fusion alone seems to be just as effective as the combination of peripheral and central fusion in maintaining straight eyes.

Apparently the monofixator has such a poor prognosis to ever become a bifixator that no therapy of the disorder appears justified other than providing the ideal optical correction and occlusion for amblyopia.

## CONCLUSIONS

The monofixation syndrome has four possible sources: (*a*) primary inability to fuse similar macular images; (*b*) secondary to treated strabismus; (*c*) secondary to anisometropia; and (*d*) secondary to a unilateral macular lesion. The monofixation syndrome is most frequently secondary to treated strabismus, and is many times more common secondary to treated esotropia than secondary to treated exotropia, presumably because of the trend of the deviation in children to become constant in esotropia but to remain intermittent in exotropia. Both the primary monofixators and those with monofixation secondary to treated congenital esotropia have an inherent inability to bifixate, a defect that may be genetically determined.

The monofixation syndrome may be present in patients whose cover-uncover and alternate cover studies reveal orthophoria, a totally compensated phoria, a partially compensated phoria with a small residual deviation, or in small totally uncompensated heterotropias. The largest cover-uncover deviation is 8 prism diopters, although the alternate cover deviation may be two to three times greater. The diagnosis of monofixation syndrome is suspected in only two-thirds of the patients by cover-uncover, since in one-third neither eye shifts with this test. The diagnosis of monofixation syndrome in patients whose eyes are within 8 prism diopters of straight by cover-uncover is confirmed by demonstrating a scotoma in the macular field of one eye and peripheral binocular single vision. The scotoma is absolute in all monofixators and facultative in all except those with an organic

unilateral macular lesion. It is demonstrable in either eye in patients who can maintain fixation of the fixation target with each eye during binocular testing.

The scotoma is demonstrated by binocular perimetry and Bagolini striated lenses and inferred by responses from testing with Worth four lights, the polaroid vectographic slide Snellen letters, the 4-diopter base-out prism test, and Wirt Stereotest stereoacuity of 67 seconds of arc or less. The most reliable of these tests are the Worth four lights and the polaroid vectographic slide Snellen letters responses, the Wirt Stereotest stereoacuity, and binocular perimetry. The Bagolini striated lens test is the most difficult and the 4-diopter base-out prism test is the least reliable.

The motor alignment in the monofixators is not the cause of the scotoma. The scotoma does not develop in response to diplopia; instead, the deviation often is reduced by the fusional vergence to use the scotoma for elimination of diplopia. The monofixation syndrome patients have good fusional vergence amplitudes, usually ample to totally compensate for the alternate cover deviation, and many partially reduce their alternate cover deviation leaving a small residual tropia by cover-uncover.

Normal retinal correspondence peripheral fusion seems to be present in the monofixation syndrome. (According to the current definition of retinal correspondence, the findings in the patients with a deviation indicate ARC, and in those without a deviation the findings indicate NRC. However, the definition of ARC is questioned.)

The monofixation syndrome frequently causes amblyopia unless the young patient alternates fixation; amblyopia is not the cause of the monofixation syndrome. The only visual deficiency experienced by the patient with the monofixation syndrome is a less refined stereopsis than that appreciated by the bifixator, a defect that is usually unnoticed. The monofixation syndrome patients are symptomless and cosmetically appear excellent.

Treatment by orthoptics, prisms, surgery, or miotics seems unable to convert the patient with monofixation to bifixation. The scotoma persists regardless of treatment. The amblyopia secondary to the monofixation syndrome is easily managed in young children by occlusion therapy. The scotoma persists despite the elimination of the amblyopia. Possibly, the prescription of anisometropic spectacles at a very young age may permit bifixation to develop rather than monofixation to persist. The prognosis that the eyes in a patient with monofixation will remain unchanged throughout life is excellent.

SUMMARY

The combination of straight or nearly straight eyes, peripheral fusion with fusional vergence amplitudes, an absolute scotoma including the macular field of the non-fixating eye, and appreciation of gross stereopsis constitute the monofixation syndrome. Various facets of this disorder have been described and called many different names, but the various categories comprising this syndrome are brought together and discussed in this paper.

One hundred consecutive patients with this syndrome were studied, and the largest deviation encountered during binocular viewing was 8 prism diopters. Thirty-seven patients had no deviation according to cover-uncover, and 25 of these patients had no deviation to alternate cover. In 26 of the 63 patients manifesting a shift by cover-uncover, the deviation was larger by prism and alternate cover than by cover-uncover (simultaneous prism and cover); 60 patients had an esotropia shift by cover test, and three had an exotropia shift.

Either the history or the visual acuity at the time the patient was examined for this study revealed that 78 patients were amblyopic; only 8 had eccentric fixation at the time of the study.

The monofixation syndrome occurred in 81 patients secondary to one or a combination of causes: (1) strabismus in 66; (2) anisometropia in 6; (3) strabismus associated with anisometropia in 8; and (4) associated with a unilateral macular lesion in 1. Nineteen of the patients were called primary since they were not secondary to any of the aforementioned factors.

The scotoma was demonstrated by the distance Worth four lights projecting within the scotoma at 20 feet, precluding fusion. By approaching the lights, the average monofixator fused them at 10 feet. The scotoma was plotted by binocular perimetry. It was facultative in every patient except the one with a unilateral macular lesion. Also, the scotoma was demonstrated with Bagolini striated lenses, polarized Snellen letters on the polaroid vectographic slide, and the 4-diopter base-out prism test. The most difficult of the various scotoma tests for the patient was the Bagolini striated lens test. The 4-diopter base-out prism test was the least reliable.

The average fusional vergence amplitudes were the same in the monofixating patients as in the bifixating patients.

The Wirt Stereotest stereoacuity was less in the monofixator (67 to 3000 seconds of arc) than in the bifixators (14 to 40 seconds of arc). The distance stereoacuity determined with the polaroid vectographic

slide correlated poorly with the Wirt Stereotest values, but did differentiate monofixation (180 seconds of arc or worse) from bifixation (60 seconds of arc or better).

Binocular perimetry requires a dissociated technique and under these circumstances invariably NRC was disclosed in all monofixators regardless of whether deviation was present or not. The Bagolini striated lenses offer a non-dissociated test circumstance and ARC was the response in those with a deviation to cover-uncover, but NRC was the response in those without deviation. These responses are determined by the retinal correspondence definition that separates NRC and ARC according to the limits of Panum's fusional space at the fixation point on the horopter. This strict interpretation of retinal correspondence would allow only 10 minutes (1/3 prism diopter) of deviation of the visual axes at the fixation point. However, the 3-degree scotoma encircling the visual axes of the deviated eye makes this strict definition of retinal correspondence meaningless, since the limits of the stretched-out Panum's fusional space peripherally to the fixation point on the horopter determines the amount the eyes may be deviated in the monofixation syndrome and still have NRC. Because some unexplainable strong driving force exists that reduces the cover-uncover deviation to within 8 prism diopters in such a large number of patients; and because these patients who have a deviation have the same normal fusional vergence amplitudes and stereopsis appreciation as those without a deviation; and because of the NRC revealed by binocular perimetry in patients with a deviation – the retinal correspondence probably is normal in all monofixators, and they enjoy the same peripheral fusion regardless of whether there is orthophoria or a deviation according to cover-uncover ranging up to 8 prism diopters.

The monofixators are symptomless, cosmetically straight, and tend to remain unchanged with increasing age. No treatment has been successful in converting them to bifixation. The amblyopia associated with monofixation is manageable with occlusion.

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